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Symposium on Adolescence

FOREWORD

The subject of adolescence is becoming of increasing interest and importance to the pediatrician. At the 1941 Academy meetings of Region I in New Haven in March, and of Region III in Chicago in May, the program committees quite independently selected the subject of adolescence for a half-day symposium. The material presented seemed to the Editors of so much interest and value that it was decided to publish the group of papers in one issue of the JOURNAL. The subject is so extensive and has so many phases that there was very little overlapping of the subjects discussed at the two meetings. Five of the papers given at New Haven and five at Chicago are included. To these a study of the metabolism has been added. The papers included in this symposium are as follows:

GENERAL CONSIDERATIONS. CERTAIN PROBLEMS OF PUBERTY AND ADOLESCENCE.

Lawrence K. Frank, New York, N. Y.

Vice-President, Josiah Macy, Jr., Foundation

SOME OBSERVATIONS ON THE GROWTH AND DEVELOPMENT OF ADOLESCENT CHILDREN

William W. Greulich, Ph.D., Cleveland, Ohio

Director of the Brush Foundation, Western Reserve University

EXAMINATION OF THE ADOLESCENT FEMALE

Joseph L. Baer, M.D., Chicago, Ill.

Professor of Obstetrics and Gynecology, Rush Medical School

MENSTRUAL ABNORMALITIES OF ADOLESCENCE

E. L. Sevringhaus, M.D., Madison, Wis.

Professor of Medicine, University of Wisconsin

ENDOCRINE PROBLEMS IN ADOLESCENCE

Ephraim Shorr, M.D., New York, N. Y.

Assistant Professor of Medicine, Cornell University Medical College

THE STANDARD METABOLISM OF ADOLESCENCE

Bruce Webster, M.D., Helen Harrington, and L. M. Wright, New York, N. Y.

Department of Medicine, New York Hospital, and Cornell University Medical College

OBESITY IN RELATION TO PUBERTY

Hilde Bruch, M.D., New York, N. Y.

Department of Pediatrics, College of Physicians and Surgeons, Columbia University

WHAT TO DO ABOUT THE FAT CHILD AT PUBERTY

Fred W. Schlutz, M.D., Chicago, Ill.

Professor of Pediatrics, University of Chicago

ATHLETIC ACTIVITY AT PUBERTY, WITH SPECIAL REFERENCE TO THE CARDIAC AND TUBERCULOUS PATIENT

Stanley Gibson, M.D., Chicago, Ill.

Professor of Pediatrics, Northwestern University

SEXUAL EDUCATION OF THE ADOLESCENT

George Mohr, M.D., Chicago, Ill.

Associate Professor, Department of Criminology, University of Illinois

PSYCHOLOGIC ASPECTS OF ADOLESCENCE

Douglas A. Thom, M.D., Boston, Mass.

Director of the Habit Clinic for Child Guidance

Dr. A. Graeme Mitchell presided at the symposium in Chicago only a fortnight before his death on June 1. His remarks at the opening and closing were informal and were not prepared or intended for publication. A stenographic transcript became available, however, and as they are so characteristic of Dr. Mitchell and his interest in and thought on adolescence, the Editors feel it only fitting that they should be included.

CHAIRMAN MITCHELL [Opening Remarks].—This particular program this morning is one which I have been told I really haven't anything to do with, that I just have to sit on the platform and smoke and be as comfortable as possible. In fact, the committee definitely warned me not to talk. That, of course, was a very tall order for me and I don't intend to obey it because I do think that some mild type of preface ought to be given to this discussion of adolescence.

As you see by the program, we are going to discuss this morning only certain phases of adolescence. In the Academy there have been from time to time discussions on the subject of adolescence and our discus-

sions have not been completed as yet and I doubt if they will be for some time to come. To me it is rather significant that we are carrying on a discussion on adolescence. The pediatrician is manifesting not only an increasing interest but also an increasing activity in the problems of the adolescent. I see no reason myself why he should not. The pediatrician has a background of interest in the subject. He is the fellow who perhaps more than anyone else is and should be interested in growth and development, and furthermore, often in the individual instance he has the background of the individual child whom he has seen through the early part of childhood, and hence I know of no one better able than he to carry on the studies and the direction of adolescence.

Adolescence is not a particularly easy period to go through as we observe it in individual instances, but most of us managed somehow to get through it or I suppose we would not be here today. In literal definition, adolescence lasts from the time of puberty until about 21 years. Today we are not discussing this whole subject, only certain phases of it and perhaps particularly that part of adolescence which we might call prepuberty and puberty. As we think of the adolescent we have to discuss the various phases of physical and emotional or mental development because all of these phases are inseparable. Our object is to get the child through childhood and through adolescence, so that eventually he can function physically and emotionally in the community, or so that he can, let's say, be ethical and be fair to the group.

I hope that what I have said you will not recognize as plagiarism, because I have deliberately taken it from something which has been written before about adolescence, but since I happened to write it myself, it is not plagiarism.

I think I ought to explain to the speakers that I am a very severe chairman and they ought to understand that from 9:15 until 12 o'clock is not six times thirty minutes. It is a little less than that. I am going to ask the speakers not to speak over twenty minutes, because while this is not a panel discussion, it is not so announced, I think some questions might come up from the audience and we should save some time for questions. So I have a mallet, and if that doesn't work I have a bomb, and I intend to hold the speakers each to twenty minutes.

[Closing Remarks.] I think that I can express the feeling of this audience in expressing our gratitude toward the speakers, particularly those who are not members of the Academy. Those who are, we expect to perform for us when we call on them, but we have had others who have contributed in extremely helpful and significant ways.

Dr. Veeder has given me permission to state to you that these papers are going to be published in the JOURNAL OF PEDIATRICS and I think we are all very happy about that. They will be combined with certain papers on adolescence given in Region I; one of the reasons for so doing,

of course, is to bring up the standard to which Region I needs to be brought up in comparison with Region III. (Laughter.)

There haven't been any questions, but it seems to me proper perhaps in just one or two minutes to see if we cannot summarize some of the ideas that we have heard today. While the speakers talked sometimes rather specifically from different angles, fundamentally we have learned that adolescence is a period through which we must all go and that somehow most of us get out of it in such a way that we can be adjusted to the community and other people and live with ourselves, as it were, or, in other words, we eventually become ethical and we learn to act properly toward the rest of the community but at the same time we retain our individuality.

There are, however, certain physical changes and psychic changes and conflicts which arise which seem somehow to be inherent in this period of adolescence. These are complicated and they are made worse if the environment is not correct, and by environment we mean family and siblings and playgrounds, all the various contacts in the community.

The problem of sex I think was discussed in such a way that we realize it is a fundamental problem of adolescence, but it is only part of development and it is not an individual problem necessarily. However, it does become more or less acute, certain phases of it, at the time of adolescence.

I think that we also learned and remember that there is such a thing as an hereditary background, and that different adolescents do have different capacities and capabilities, both physical and mental, and there are wide limits to what can be considered normal; that is, normality is not a line or a series of spots on a chart, and we must be careful to see that the child is not pushed either physically or intellectually beyond his individual capacity. Somehow we have to determine what his individual capacity may be. We do not want too much standardization in the schools and an attempt to make all children absorb the same information and do the same things in the same length of time because I think we liked the discussion of the uniqueness of each individual. I think it is a very good point for us to remember.

I think we must not expect the educator to correct the background for which he is not responsible. I think we are all aware of the deficiencies in the relationship of the physician to the school and we are thinking about such problems and we must do something to solve them. The plea was made that we have more joint action. After all, some pediatricians at least go to joints and I see no reason why we should not have more joint action with the educators. (Laughter.)

I think also in discussing growth we came back to this individuality again. Some children are going to be tall and some short and some are going to be more or less round, and some are going to be narrow. These characteristics are fundamental and cannot be changed, and often we

ought to be careful not to attempt to change them. We recognize the physical individuality of the child as well as the psychic and intellectual individuality. There are broad bands here also as to what constitutes normality and what constitutes the proper time for the beginning of puberty. It requires individual study to find out whether the child is normal.

I think we came to the conclusion, which we had before, that as pediatricians we have a function to perform in the guidance of the physical and mental care of the adolescent, and it is a function which perhaps we can fulfill in many respects at least as well as anyone else and perhaps better. We had better carry on this particular job and ask for assistance when we get into difficulties which are too complex for us to solve. We may need the endocrinologist from time to time, the gynecologist, and the educator on a cooperative job with the pediatrician as a guide, and certainly we can no longer refuse to give our assistance and our knowledge with our background to the child who is over 12 years of age; that is, the adolescent belongs to us.

I think that we got out of this also that the care of the child is an individual matter, although he is subjected to mass procedure to which he must adjust, and the manner in which he adjusts to these mass procedures and to the community will depend upon his individual background.

GENERAL CONSIDERATIONS
CERTAIN PROBLEMS OF PUBERTY AND ADOLESCENCE

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IN OPENING the discussion, it may be appropriate to review some of the larger aspects of this topic in the hope that we may provide a perspective through which the more specific presentations may be seen.

Twenty years ago the preschool child was revealed as the forgotten, almost unknown child. During the 1920's and early 1930's many studies were made of the preschool child which have thrown much light on that age period. Today we are realizing that the adolescent is the forgotten child about whom little is known and for whom little systematic care is being provided. The customary practice of limiting pediatrics to the period before puberty has resulted in a sort of interregnum or no man's land for the years between puberty and the time when the internist begins to see the late adolescent or young adult. Perhaps nothing is more encouraging than the inclination now being shown by pediatrics to extend its interests throughout the second decade, although this arrangement may encounter some difficulties with respect to hospital care.

Let us first look at the broad statistical picture so that we will have before us some conspectus of the adolescent population. While the number of infants and young children is decreasing fairly rapidly, as shown by an absolute decrease of over 300,000 children under 5 years of age in the 1940 census, a shrinkage in the adolescent population will probably not occur for another decade or so.

It is worth noting that the lowest death rate for all age groups occurs in the age period of 10 to 14 years, but that in the next age period, 15 to 19 years, there is a very sharp rise toward the adult death rate.

It is also significant that the female death rate, which is consistently lower than the male death rate, until very recently has been higher than the male death rate in this age period of 15 to 19 years. The decline in the death rate for the age period of 10 to 14 years from 1911 to 1938 was less than in any other 5-year age period from 1 to 34 years. The decline in the death rate for the age period of 15 to 19 years was only slightly better, indicating that the second decade has not profited from medical advances as much as infancy or even early adult age during the past twenty-odd years. The incidence of physical defects and handi-

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caps among the draftees may also be cited as indicating the neglect of adolescents. Opinions will differ as to whether the sudden rise in mortality from 15 to 19 years of age is to be attributed primarily to the stress and strain and hazards of early adult life or to the neglect of childhood and especially of the age period of 10 to 14 years.

For the past eight or ten years, several intensive studies of growth, development, and maturation of boys and girls during the second decade have been under way.* While none of these investigations have yet appeared in definitive publications, some of the major findings are beginning to emerge. Consideration of these may be helpful therefore in illuminating the hitherto obscure second decade of life. It is desirable that we try to get a clearer understanding of what puberty and adolescence mean. In many discussions they are used more or less interchangeably and are sometimes intended to designate a particular event or epoch in the life of the individual, and at other times they refer to a process. It appears that toward the end of the first decade, that is, somewhere around 9 or 10 years of age, the developmental processes begin, or, more correctly, are accelerated, which lead to the event that we call puberty or the first stage of sexual maturation. What needs to be emphasized especially is that during these years after 9 or 10 years of age, a series of developmental changes are taking place in the skeleton, in the gonads, and in the reproductive organs, which are of considerable significance for the individual's future health and normal functioning. Because we have focused our attention primarily upon the specific event of puberty, such as the menarche, these highly important developmental and maturational processes in the preceding years have been either neglected or ignored.

It has also been customary to think of puberty as occurring at a fairly definite time period, such as 13½ years in girls, or 14½ years in boys, without realizing sufficiently that there is a very wide range, so that the menarche may occur anywhere from 10½ years to 17 years in girls, and puberty may occur in boys anywhere from 12 years to 17 or 18 years.

If we distinguish this period preceding the menarche or the first stages in maturation of the male as the puberal period, then the period following might be regarded as the period of adolescence, which would extend on into the end of the second decade or perhaps even into the third decade. During this period of adolescence, the process of sexual maturation continues as the individual moves on toward maturity. Here again it should be recognized that the menarche represents only the first stage of sexual maturation in the female and that the later stage of actual ovulation may not occur for one, two, or even three years following the menarche. In this interval it has been pointed out that the female organism has to learn the specific sexual rhythm that is

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appropriate to her individual organism, and therefore it is more than appropriate to speak of a "normal menstrual irregularity," to use the very effective phrase of Dr. Leslie B. Arey.

Studies of what takes place during the second decade suggest that we may gain an understanding of the processes of puberty and of adolescence if we recognize that there is a more or less orderly, sequential process of growth, development, and maturation, but that each individual boy and girl will pass through this sequence (a) at his or her own rate of progress, and (b) with structural and functional dimensions that are peculiar to his or her constitution, heredity, etc. Consider 100 boys and 100 girls, traveling from New York to Chicago, some by airplane, some by Twentieth Century Limited, others by bus, some by water, some by hitch-hiking, and others trudging along on foot. Those who get to Chicago will arrive at different times and in different conditions, depending upon their mode and rate of travel. Each route will impose different costs upon the individuals traveling that way. This indicates that to understand or evaluate adolescence it is important to study the process and the rate at which the specific individual is moving rather than to rely upon chronologic age norms or standardized tables of height, weight, and other bodily dimensions. This becomes clearer when we realize that the process of maturation is not necessarily correlated with growth in external dimensions. Indeed, one may hazard the generalization, for which there is an increased body of evidence, that the shorter and perhaps broader girls mature sexually earlier than the tall, thin girls, and this probably also applies to many boys. There has been considerable confusion on this point because of a very easily understood confusion of language. Various studies have indicated that the tall girl reaches the menarche earlier than the shorter girl which is true to the extent that the girl who is taller at eleven or twelve will reach puberty earlier, but such early maturing girls will not attain the same terminal stature as the girls who reach puberty much later, since this delay in sexual maturation permits continued growth, especially in the extremities, and therefore leads to a greater terminal stature.

One aspect of the adolescent development that is to be noted especially is the difference in these growth patterns. Some boys, for example, about 14 or 15 years of age will grow very rapidly for a period of sixteen or eighteen months and attain almost their full adult stature within this short period of time. Another group of boys will grow fairly slowly over a longer interval and attain almost equal stature by the end of the second decade.

In these growth patterns we can see a contrast which may have a very large significance for the vitality and health of the individual boy. In the first case we may see a boy with adult stature but with a cardiovascular and a gastrointestinal system, muscular development, gonadal

development, and a degree of emotional maturation that are still juvenile.

This points to an important observation that is beginning to emerge; namely, that adolescence may be a period of organic incongruity when the individual child must attempt to live with organ systems and functional capacities of widely varying levels of maturity and functional efficiency. These organic incongruities or discrepancies may be a serious hazard to the individual if, as so frequently happens, he or she attempts to live up to this most advanced maturity, such as stature; or if the individual is called upon by parents, teachers, and others to live according to his or her most advanced development. It is suggested that both chronologic age and stature, which have heretofore been used as a basis for grouping children or for assigning tasks and activities, may work a great hardship on the individual child who does not conform to these structural norms or averages.

If we view adolescence as a period of rapid growth and of organic imbalance, then we may gain some clue to an understanding of the frequently puzzling and often disconcerting behavior and functioning of adolescents. Whenever we have an organism that is rapidly growing, especially when growing in an asymmetrical or unbalanced manner, it is likely that such an organism will be physiologically unstable; the very change in dimensions through which growth takes place precludes any steady, stable functioning. Moreover, if we examine the implications of the growth process taking place during puberty and adolescence, we may gain some clues to the many defects, maladjustments, instabilities, and difficulties exhibited by boys and girls during the second decade. For example, it may be pointed out that the adolescent who is rapidly increasing his stature in a very short space of time may suffer from a deficiency of vitamins and minerals necessary to sustain that rapid growth, thus being in a physiologic condition that may be called "adolescent rickets." Moreover, it seems clear that the enlargement of the skeleton that occurs in these rapidly growing individuals will be accompanied by a period of muscular incoordination, since all the neuromuscular adjustments of childhood will be disrupted and the individual must build up a new set of eye, hand, etc., coordinations commensurate with his suddenly enlarged organism. During later adolescence the individual boy and girl will attain the more or less "steady state" of maturity, depending upon how well they develop and organize the physiologic process of homeostasis. It seems probable that some of the organic imbalances or physiologic instabilities of adolescence may persist on into the adult years, where they become, so to speak, frozen, thereby giving rise to persistent physiologic imbalances and functional inefficiencies that constitute a definite handicap, if not hazard, to adult health and full functioning.

It is customary to hear parents and other adults speak of the adolescent child as a continual source of worry, anxiety, and irritation, but it is not so often recognized that the adolescent himself may be in a condition of chronic confusion and dismay arising from his own changing body and its organic instability, his own incoordination, and the difficulty of meeting the many and onerous burdens that are laid upon him by parents and schools. It should also be remembered that the process of sexual maturation involves not only a series of internal, organic developments, but likewise brings to the boy and girl a series of very perplexing and sometimes highly disturbing emotional problems and social difficulties. It has been frequently pointed out, for example, that adolescents are inclined to be especially anxious about their normality. Whatever their size or their stage of maturation they will compare it unfavorably with their chronologic age group and worry lest they are too large, or too small, whether they are precocious or retarded, whether one portion of their anatomy, especially the genitals, is of the right size, shape, and functional efficiency. Again it must be realized that with the enormous emphasis placed upon appearance and "good looks" of the individual, many boys and girls will undergo a period of considerable stress and strain over their inability to conform to the prevailing or more fashionable type which in many cases may be completely inappropriate to their constitution, heredity, and other individual characteristics. It is important to emphasize this anxiety over normality on the part of adolescent boys and girls, because it frequently complicates the physician's task. First, the adolescent may be very reluctant to acknowledge any disorder or malfunctioning just because it is a source of anxiety which he fears may be confirmed by medical examination. Second, because his or her own organism and its functioning are so frequently invested with strong emotional significance, as something that one should not talk about or think about, as something that is low and nasty or unclean, we again have a situation in which the adolescent may be strongly resistant to the medical or health teachings that are offered him. It should also be remembered that since the parents of adolescents are frequently very solicitous about their children's health, welfare, and conduct, their well-meant efforts to guide their adolescent children and to provide the right regime of food, sleep, and living again may be strongly resisted; the adolescent is concerned primarily with the question of conforming to the requirements of his own age group and of achieving some measure of independence from his parents as a first step toward the attainment of adult living.

Perhaps it is not unwarranted to say that the growth, development, and maturation that take place during the second decade may be viewed as basic biologic processes, but for the individual boy and girl undergoing those processes they are the focus of a great many cultural, social,

family, and emotional concerns that make them much more than purely biologic processes.

If, as indicated earlier, we regard the second decade as a period of transition from infancy to adult maturity and recognize that each individual girl and boy will traverse this period at his or her own rate of progress and will exhibit a pattern of development that is highly idiomatic or idiosyncratic, then it would appear that pediatrics is faced with a very large opportunity and a most important responsibility in providing an adequate program of health care during this second decade of living.

Until very recently it has been customary to think of health and sanity as something that is lost through some mysterious process which is difficult to explain. Studies of child growth and development, especially the clinical studies, make it increasingly clear that health and sanity have to be achieved by the individual by meeting the tasks of life and the demands of maturation without impairing the integrity of the organism and of the personality.

We cannot expect the individual boy and girl to meet these requirements effectively if in our various programs and practices in the home, in the school, the clinic and hospital, and in the youth organizations and elsewhere, we persistently deny the basic, ineradicable differences among individuals and impose upon them the tyranny of the norm. Perhaps in no other period except old age is there a greater need for the clinical approach which explicitly recognizes this individuality and mediates both the requirements and the needs of adolescents in accordance with the peculiar make-up and capacities of the individual boy and girl.

This points then to some very difficult questions of how pediatrics, if it is to accept its responsibility in the second decade, can effectively contribute to the better nurture and care of adolescents. It is believed that medicine has much more to contribute to education than routine physical examinations and correction of defects, however important these may be. Schools, camps, youth organizations, all the different programs through which children pass, are all in need of the guidance and illumination which medicine, especially pediatrics, can bring to bear upon those programs. For example, we have the situation now in which the boys and girls of 10 to 13 or 14 years, who are undergoing these highly significant puberal changes, are usually under the greatest pressure in schools in getting ready to graduate from the elementary and move into the high school. Indeed, it might be suggested that if pediatricians were to examine the school and other youth programs during the second decade from the viewpoint of what they are doing to and for adolescents, they might find many occasions for protesting against requirements and practices which violate the integrity of the developing organism and only increase the difficulties of achieving a wholesome, sane maturity. The question may be raised as to whether the

place and function of the physician in the schools does not need to be reconsidered so that he can become the agent through which the knowledge of growth, development, and maturation and, more especially, his clinical knowledge and training, can be focused upon the schools, buildings, equipment, programs, and activities. In this connection one cannot help but wonder whether the preoccupation with the diagnosis and correction of defects, which has been so much stressed as the medical contribution to education, may not be obstructing the more important problem of preventive medicine. For the adolescent years medicine would seem to be not so much a matter of diagnosis and therapy as the question of providing a regimen of nutrition, rest, activity, and release which will help the individual boy and girl to achieve maturity with a better organic balance and coordination and with a functional efficiency appropriate to his specific organism.

Just because of the adolescent's attitude toward the home and family, it may be necessary to focus the efforts of health care of adolescents through the schools or other adolescent groups, wherein some of the strong desire to be like everyone else can be utilized to advance the program of health care. Everyone knows how difficult it frequently is to persuade an individual boy or girl to modify his diet or activities when approached as an individual, as contrasted with the alacrity with which he will enter upon even a most rigid program of diet, exercise, and regularity of activities when presented as a group or team requirement. Therefore, instead of working the hard way, perhaps we should make greater use of this propensity toward group activities. Here at this point it may be appropriate also to suggest that, while there have been few careful studies made, there are some indications that many of the psychosmatic disorders which are found among adults may have their origin or their period of fixation during adolescence. It is enough to remind you that often the adolescent girl may utilize headaches, malaise, or her menstrual function as an outlet for gaining attention or evading some requirements or the demands of life. As individual boys and girls are physiologically unstable and are also subject to considerable stress and emotional strain, as so many are, it is not difficult to see how those emotional problems may become focused upon some organ system or function as a mode of release, which, if it serves effectively to protect the individual, may then become a persistent dysfunction.

As indicated earlier, the number of infants and young children is rapidly decreasing and within the next decade or so we will begin to see a decline in the number of adolescents. In so far as the pediatrician is increasingly able to continue the supervision of the child from infancy on, it would seem highly appropriate for him to continue his professional direction through the second decade. Much of the mystery and obscurity that has surrounded the second decade will be dispelled by the studies that are now being made on adolescent development, so

that the pediatrician can enter upon this problem with increasing confidence that his clinical training, supplemented by the growing knowledge of adolescent development, will enable him to render a much needed service in the protection and conservation of our diminishing child population, not merely through childhood but on through early maturity. While it would be premature to assert any confident predictions, it seems highly probable that within the next decade at the least we will have to work out a fundamental policy and program of health care for childhood and adolescence. It is hoped that the pediatricians will be ready and willing to assume their full share of responsibility in approaching this vital task.

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See also Child Development Monographs for reports on adolescent studies.

SOME OBSERVATIONS ON THE GROWTH AND DEVELOPMENT OF ADOLESCENT CHILDREN

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THE normal human life span may be considered as divisible roughly into three epochs: the first, which extends from conception until well into the third decade, is the time required to attain adulthood; the second is the variable, but usually very brief, period during which the individual enjoys the peak of his physical efficiency; and the third is the period of physical deterioration, a process which begins insidiously as early as the late twenties and, gathering speed as the years go by, effects his eventual senility and dissolution. Viewed in this way, it appears that the first twenty-odd years of life are spent in achieving physical maturity and a degree of physiologic equilibrium which is lost, at least in part, almost as soon as it is attained. If this seems an exaggerated statement, I would remind you of the common observation that the baseball player is usually well past the peak of his physical efficiency at 30 and that the pugilist's legs have already begun to slow him down some years earlier. Viewed in this way, it is, perhaps, not too much to say that man has scarcely begun to live when he begins to die.

We need not dwell longer on this morbid note, nor need it unduly distress us; for, after all, there are still some pleasures left in life after most of the agility of youth has been lost. I give you this concept merely because it serves to put into a somewhat clearer perspective that particular period of life, adolescence, which I wish to discuss with you.

Adolescence begins early in the second decade and is usually considered as ending at about the twenty-second or twenty-third year in boys and somewhat earlier in girls. It covers, therefore, approximately the last ten years of what I have just referred to as the first epoch of life, the period extending from conception to adulthood. The physical changes which occur during this early period of life involve both growth and development: growth, in the sense of an increase in mass, volume, and external dimensions; and development, in the sense of becoming progressively more complex. Now, these two processes, growth and development, do not proceed at the same absolute rate or at the same relative rate throughout this early period of life. There are intervals during which the body is increasing in size more rapidly than it is growing in complexity, and there are other times at which this relationship is reversed. The physical changes which occur during adolescence are, perhaps, best appreciated when viewed in the light of some events which have preceded them. Let us, therefore, consider briefly some of

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the more conspicuous physical changes which have occurred in the child by the time adolescence begins.

I want to digress at this point to tell you a story. A kindergarten teacher whose class had spent some days drawing and cutting out pictures of various animals decided to test the children's powers of observation. She asked her class this question: "What do elephants have that no other animals have?" The little girl who volunteered to answer the question did not say, "A trunk," as the teacher had hoped she would, but instead replied, "Little elephants." The child was wise beyond her years, for she had hit upon an aspect of the matter which is so obvious that it usually escapes her elders.

When we speak of things which are determined by heredity in the human body, such traits as eye color, hair color, hair form, skin color, or such defects as hemophilia, red-green color blindness, etc., suggest themselves. We are likely to lose sight of the fact that, in addition to determining a vast number of what may more or less properly be called unit characters, such as those I have just enumerated, there is also in the germ plasm some mechanism which controls larger aspects of development and which insures, for example, that human beings give rise only to other human beings and that elephants continue to produce only elephants.

The genetic constitution of man, as well as that of other forms, controls another important aspect of development. Not only does it determine within rather narrow limits what the end product of development will be, but it also prescribes quite definitely the stages to be followed in attaining that end. One expression of such genetic control is seen in the human growth pattern, a consideration of which will return us to the principal theme of this paper.

Most of the existing information on the human growth pattern is based on what has been termed cross-sectional data which take the form of average heights and average weights of boys and of girls of various chronologic ages. Such values are usually obtained in this way: An investigator measures and weighs a reasonably large number of boys and girls of, say, 6 years of age, and determines the average height for each sex. He then measures and weighs an approximately equal number of boys and girls of 7 years of age and determines the corresponding average values for them. The same procedure is used for other age groups, and the investigator eventually emerges with figures, which, when translated into tables and graphs, are very impressive and are rather generally accepted as a faithful delineation of the human growth pattern. Data of this kind have proved to be of considerable practical value. They suffer, however, from certain inherent disadvantages; and I shall call attention to some of those disadvantages as we proceed. Let us consider first an example of a pattern of human growth which is based on such cross-sectional data.

Fig. 1 shows graphically the average standing heights of boys and girls for each year from birth to 20 years of age. The average heights of the boys are shown in solid black, while those of the girls are repre-

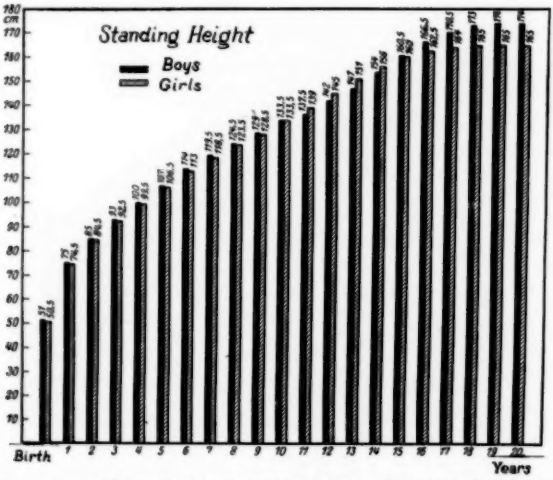

Fig. 1.—Average standing heights (in centimeters) of boys and girls from birth to 20 years of age. (From Peter, Wetzel, and Heiderich: Handbuch der Anatomie des Kindes, Bergmann.)

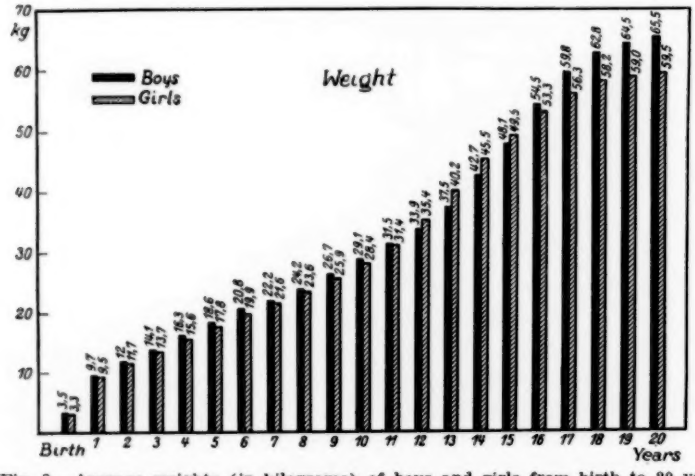

Fig. 2.—Average weights (in kilograms) of boys and girls from birth to 20 years of age. (From Peter, Wetzel, and Heiderich: Handbuch der Anatomie des Kindes, Bergmann.)

sented by crosshatching. This figure is taken from Pfuhl's (1928) study of South German children. I have chosen these German data rather than comparable data from this country because they are based on a somewhat more homogeneous population than our own. The observed average standing heights for the boys and for the girls are

recorded separately for each age group. It will be noted that boys are somewhat taller (longer) than girls at birth and that they maintain this superiority up to about the age of 10 years, at which time the girls equal the boys in stature. From the ages of 11 to 14 years the girls are taller than the boys, but by the age of 15 years the boys have grown slightly taller than the girls. The boys' superiority in stature increases during the next four years and is maintained thereafter.

Fig. 2, from the same source, shows the average weight in kilograms of the same group of boys and girls from birth to 20 years of age. It will be noted that whereas at 11 years of age the average height of girls is about 1.5 cm. greater than that of the boys, the boys still remain slightly heavier than the girls. The reverse is true at the age of 15 years, at which time the boys again exceed the girls in height, although they are still surpassed by them in weight.

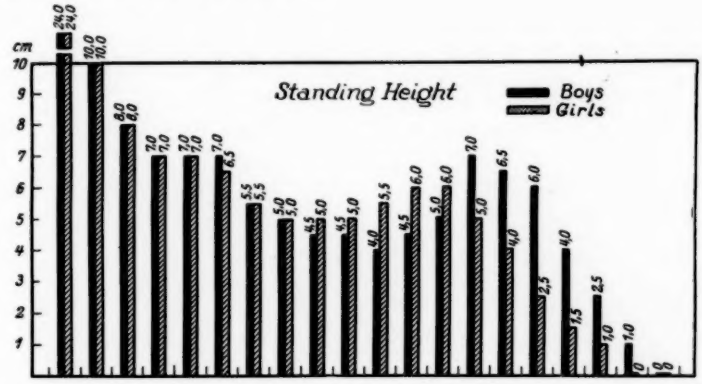

Fig. 3*a*.—Average increments in standing height of boys and girls from the first through the twentieth year. (From Peter, Wetzel, and Heiderich: Handbuch der Anatomie der Kindes, Bergmann.)

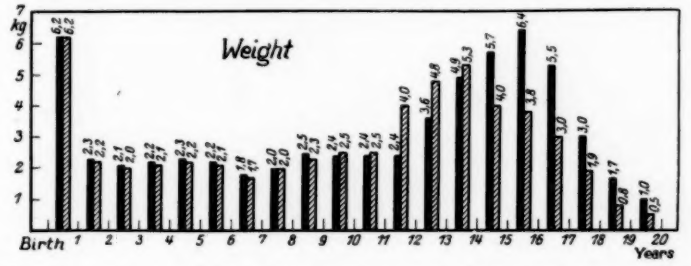

Fig. 3*b*.—Average increments in weight of boys and girls from the first through the twentieth year. (From Peter, Wetzel, and Heiderich: Handbuch der Anatomie der Kindes, Bergmann.)

The pattern of growth is shown somewhat more clearly in Fig. 3, in which the average annual increments in height and weight, rather than the absolute heights and the absolute weights, are represented graph-

ically. Average heights and average weights alone, of course, give no information about the range of variability of these two aspects of growth in the group from which the data were derived. Such averages, therefore, tend to obscure differences which become apparent when one compares the growth pattern of individual children. A study of individual growth curves discloses, for example, that all adolescent boys do not have their maximum spurt of growth at the same age; and that girls also show similar differences. This fact is well illustrated in Figs. 4 and 5, from Shuttleworth's (1939) monograph, which is based on the data from the Harvard Growth Study.

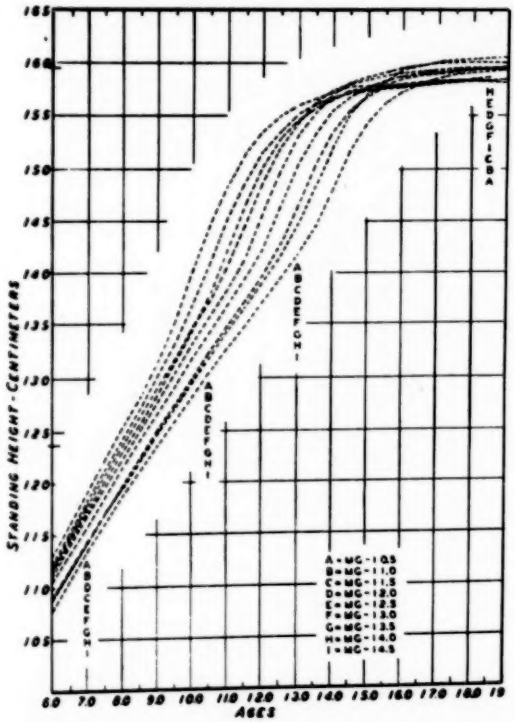

Fig. 4.—Growth trends in average standing height of nine groups of girls having different ages at maximum growth. (From Shuttleworth: Monographs of the Society for Research in Child Development, vol. 4, No. 3, National Research Council, 1939.)

Fig. 4 depicts growth trends in average standing height of nine groups of girls having different ages at maximum growth. Fig. 5 shows similar differences in ten groups of boys. It will be noted that there is a considerable range in the average standing height of the girls between about 10 and 14 years of age and a somewhat comparable range in the average standing height of the boys between about 13 and 17 years of age. Ob-

viously, a single average height for either the boys or for the girls at any one age within those periods would be much less representative of the group than would their average heights at 7 or 8 years of age, when there is a much smaller range in their stature.

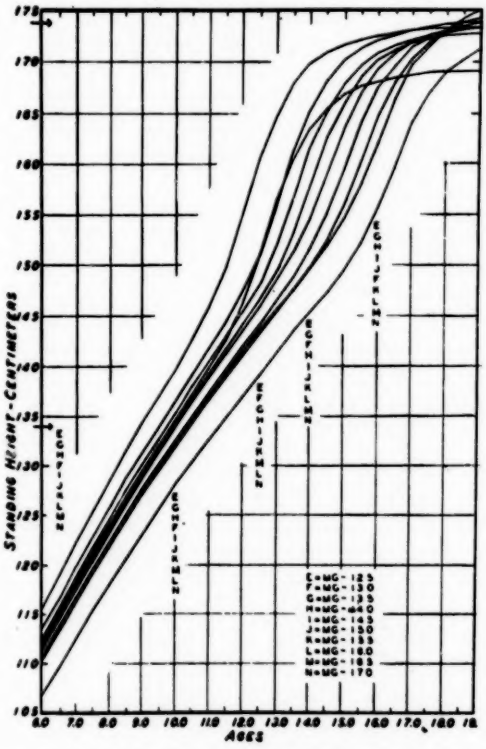

Fig. 5.—Growth trends in average standing height of ten groups of boys having different ages at maximum growth. (From Shuttleworth: Monographs of the Society for Research in Child Development, vol. 4, No. 3, National Research Council, 1939.)

We have seen an example of a growth pattern based on average increments in height and weight as observed in groups of children of different ages. It should be emphasized that these were different children in each age group, not the same children weighed and measured on successive years. We have seen, also, that there is considerable variation as regards the age at which the so-called preadolescent spurt of growth occurs in different children and that the resulting variability must be taken into account when interpreting reported average heights and average weights of children in that age group.

Since such variability occurs even among children of the same racial stock, it is not surprising to find an even greater variability among adolescent children in a population as heterogeneous in race and in

national antecedents as that of many parts of our country. These facts suggest some of the limitations of the various height-weight-age standards, especially when they are applied to our adolescent children.

We have been considering the growth of children, specifically the amount that they increase in height and weight from year to year. Let us turn now to a consideration of those developmental changes which begin to manifest themselves early in the second decade of life and which have come to be recognized as representing successive stages in the child's progress towards maturity.

These maturative changes are believed to be initiated and, in part, maintained by hormones produced by the anterior lobe of the pituitary gland. Two of those hormones are of especial interest in this connection. The first is the growth hormone, which enables the healthy, adequately nourished child to attain its maximum normal body size. In the absence of an adequate amount of this hormone, normal growth does not occur, and a form of pituitary dwarfism results. If an excessive amount of growth hormone is produced by the pituitary gland of a child or a young adult, pituitary gigantism ensues.

The second pituitary hormone of importance in maturation is the gonad-stimulating, or gonadotrophic, hormone. In children, just before the onset of puberty, this hormone begins to be produced in a quantity sufficient to cause their immature gonads to grow and eventually to develop into mature ovaries or testes. This same hormone helps to sustain the functional integrity of the ovaries and testes throughout the individual's reproductive life. An inadequate production of gonadotrophic hormone in the preadolescent child prevents the normal growth and development of the ovaries or testes, and, indirectly, that of the other reproductive organs as well, and results in a hypogonadal or hypogenital condition in which the reproductive organs remain in an almost infantile state and the various secondary sexual characters fail to develop properly. The presence of an excessive amount of this gonadotrophic hormone in early life produces one type of precocious sexual development.

Once the gonads have begun to develop in response to the gonadotrophic hormone of the pituitary gland, they begin to produce hormones of their own in significant quantities: the ovary produces the so-called female sex hormone, theelin or estrone, and later, corpus luteum hormone, progesterone; the testes produce the so-called male hormone, probably in the form of testosterone. The female hormone stimulates the growth of the breasts, the mammary glands, the uterus, the Fallopian tubes, the vagina, and various secondary sexual characters proper to the female. The male hormone similarly stimulates the growth and development of the prostate gland, the seminal vesicles, the penis and associated structures, as well as the male secondary sexual characters, the growth of the beard, deepening of the voice, etc.

Both male and female hormones have another important function: they act upon the pituitary gland and seem to cause a gradual reduction in either the amount or the effectiveness of the growth hormone produced by that gland and eventually stop its action completely. We noted previously that the pituitary gland must produce an adequate amount of growth hormone at the proper time if the child is to grow normally.

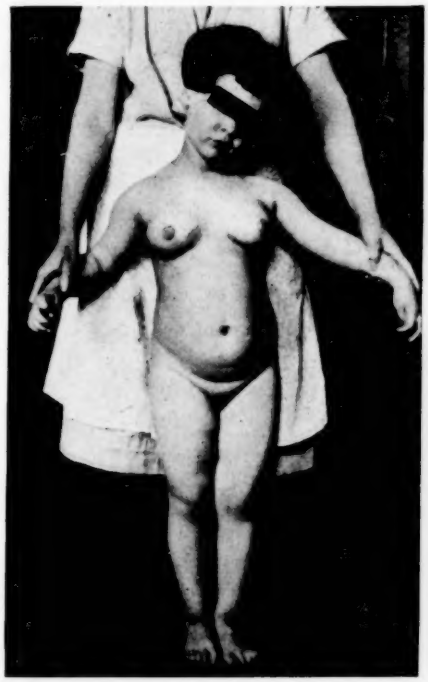

Fig. 6.—Thoms and Hershman's patient with precocious sexual development at the age of 3 years, 11 months. (From Thoms and Hershman: Am. J. Obst. & Gynec. 6: 349, 1923.)

It is equally true that the child's gonads, the testes or ovaries, as the case may be, must produce a sufficient quantity of their own hormones if growth is to cease at the proper time. If the testes or ovaries begin to function at the requisite level too early in life, growth is arrested prematurely and the child ends up abnormally short. If, on the other hand, the adequate production of the ovarian and testicular hormones is unduly delayed, growth, particularly that of the limbs, continues for too long a period and the characteristic bodily proportions of the eunuch are attained. It appears, therefore, that normal growth and development are contingent upon the reciprocal and properly timed action of pituitary and gonadal hormones. The validity of this hypothesis has

been conclusively verified by numerous experiments on animals. In this, as in so many other cases, nature has anticipated man's experiments by making some of its own on man. Let us look at the results of one of nature's tragic experiments in this field.

In 1923, Thoms and Hershman described a case of sexual precocity in a girl 3 years, 11 months of age. When she was about 1 year old, it had been noticed that her breasts were larger than normal, and when she was 3 years and 7 months old, she menstruated for the first time. Fig. 6 shows the appearance of this child at 3 years, 11 months of age. At the time that this photograph was made she was 42 inches high and weighed 50 pounds. Her physical appearance was described as follows:

"The physical examination of the head, neck, chest, abdomen and extremities reveals nothing abnormal. The breasts are those of a woman. The primary areola is well differentiated, the nipples well developed and protuberant. There is no secretion. There is no axillary hair. The pubic hair is moderately thick and covers the pubes. The external genitalia show the labia to be well developed. No internal examination has been made." X-ray examination of the wrists disclosed a degree of skeletal development several years in advance of her chronologic age.

Fig. 7[3a] shows the same child when she was 8½ years old. It will be noted that at that time she had the physical development and bodily configuration of a mature woman. She died in her nineteenth year, following the second operation for the removal of a cerebellar tumor. The photograph reproduced in Fig. 8 was made several months before her death.

During normal puberty, the development of the breasts is among the earliest external manifestations of beginning sexual maturation, and the breast changes are usually well under way before the pubic hair appears in any considerable quantity. The growth of axillary hair usually begins after the pubic hair is fairly well developed and is ordinarily preceded by the menarche, the first menstruation. It is interesting to note that, although in precocious puberty all of these changes occur abnormally early, the order in which they appear is substantially the same as that observed in normal puberty.

Children in whom puberty occurs precociously end up small in stature. This is understandable in the light of what was said earlier in this paper about the action of the gonadal hormones in suppressing the production of pituitary growth hormone. In these cases the gonadal hormones are produced abnormally early in quantities sufficient to suppress growth before the normal adult stature has been attained. It was also mentioned earlier that the absence of adequately functioning gonads during childhood results in a lack of sufficient gonadal hormones to suppress the production or the effectiveness of the pituitary growth hormone. Under such conditions, growth continues for an abnormally long period and

the individual ends up quite tall, with disproportionately long limbs and with other typically eunuchoidal bodily proportions.

Fig. 7.

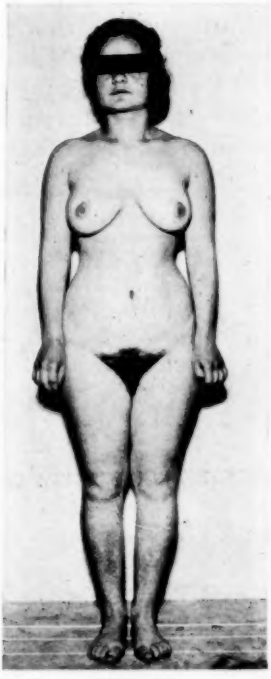

Fig. 8.

Fig. 7.—Thoms and Hershman's patient with precocious sexual development at the age of 8 years, 6 months. (Courtesy of Dr. Arnold Gesell, Yale University Clinic of Child Development.)

Fig. 8.—Thoms and Hershman's patient with precocious sexual development at the age of 18 years. (Courtesy of Dr. William J. German, Department of Surgery, Yale University School of Medicine.)

We have considered the effects of gonadal hormones on physical growth and development in two contrasting pathologic states. It should not be implied that the gonadal and pituitary hormones are the only factors involved in the processes of growth and development. If the latter are to proceed normally, however, it is essential that those hormones be present in adequate quantities at the proper time. Let us consider, next, the influence of these hormones on the growth and development of normal girls and boys.

Fig. 9 from Shuttleworth's (1937) monograph shows individual growth curves of ten girls whose first menstruation occurred between the ages of 10 years, 7 months, and 11 years, 3 months, and of 10 girls whose first menstruation occurred between the ages of 14 years, 5 months, and 15 years, 2 months. I wish to direct attention to several points

which are illustrated in Fig. 9. It will be noted, first, that the girls who attained menarche at the younger age were, on the average, taller during the period from about 8½ to 12 years than were those in whom the menarche occurred later. The girls in the late menarcheal group, however, continued to grow after the growth of the girls in the other group had practically ceased, and they ended up taller than the others. These curves show also that the rate of growth in stature decelerates rapidly after the menarche. The menarche indicates that the girl has begun her

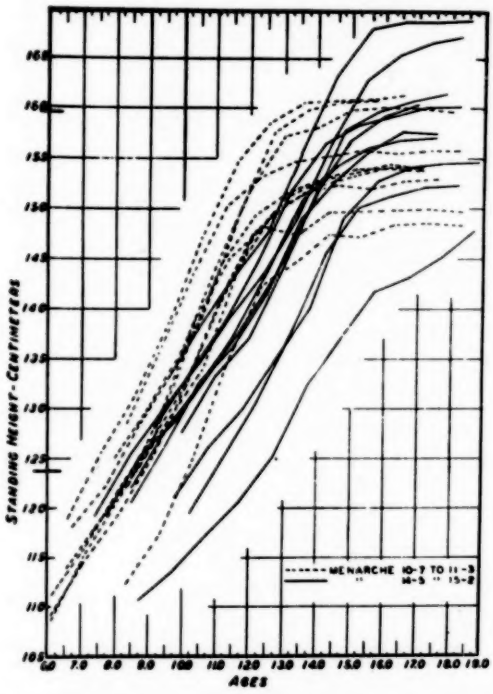

Fig. 9.—Individual growth curves in standing height, plotted to exact ages, of ten cases in which the menarche occurred between the ages 10 years, 7 months, and 11 years, 3 months (broken lines) and ten cases between the ages of 14 years, 5 months, and 15 years, 2 months (solid lines). (From Shuttleworth: Monographs of the Socie.y for Research in Child Development, vol. 2, No. 5, National Research Council, 1937.)

progress toward sexual maturity and that the quantity of ovarian and other hormones in her blood has increased to a point at which the necessary changes in the uterus which precede and accompany menstruation are evoked. Once that level of ovarian activity has been attained, the growth-stimulating action of the pituitary gland is diminished and the rate of growth rapidly decreases. This sequence of events is the same as that which occurred in the case of precocious puberty described above. That girl was quite large for her age before her first menstruation, but

she ended up very small in stature because the gonadal and other changes associated with her sexual maturation were initiated and completed abnormally early. The relation between the menarche and the deceleration of the growth curve is strikingly shown in Fig. 10, which is based on annual increments in standing height of eight groups of girls who attained menarche at different ages. The curves are arranged so that the points corresponding to the age at menarche are in the same vertical plane. Though there is no clear-cut point in the development of boys which corresponds to the menarche, they, too, show a rapid deceleration in growth after their sexual development is well under way.

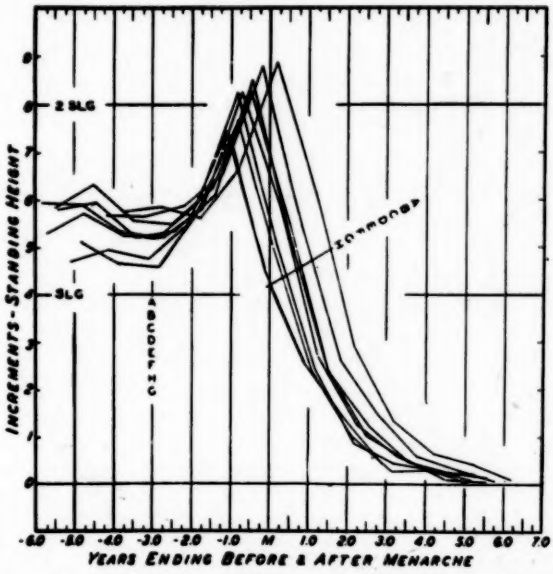
INCREMENTS - STANDING HEIGHT
2 SLG
SLG
A B C D E F G
MONTHS
-6.0 -5.0 -4.0 -3.0 -2.0 -1.0 M 1.0 2.0 3.0 4.0 5.0 6.0 7.0
YEARS ENDING BEFORE & AFTER MENARCHE

Fig. 10.—Annual increments in standing height of eight groups of girls who attained menarche at different ages. The curves are arranged so that the points corresponding to the menarcheal age are on the same vertical line. (From Shuttleworth: Monographs of the Society for Research in Child Development, vol. 2, No. 5, National Research Council, 1937.)

It might be well, at this point, to discuss briefly the rather widespread belief that the menarche necessarily indicates the attainment of a degree of sexual maturity which makes reproduction possible. It is probably incorrect. Hartman (1931) has shown that in the puberal Macaque monkey the proportion of ovulatory as compared with nonovulatory cycles is very low and that in that animal approximately one year elapses between the first menses and the first conception, despite frequent matings in the interim. Yerkes (1935) reported that a chimpanzee in his colony did not become pregnant until about thirteen months after her first menses, although she had been caged with a fertile, adult male continuously throughout that period. To those of you who are familiar with

the habits of the male chimpanzee this observation will seem especially convincing.

Evidence of a similar lack of the degree of maturity necessary for conception in puberal mice has been advanced by Mirskaia and Crew (1930). That the menarche may be associated with a comparable degree of sexual immaturity in girls in suggested by the fact that in some primitive societies in which rather promiscuous sexual relations are permitted among adolescents, the girls involved are said rarely to become pregnant during that period (Malinowski [1929] and Ashley-Montagu [1939]). Although this anthropologic observation has only the value of indirect evidence, it is consistent with what is known of this phenomenon, adolescent sterility, in other primates. In view of our present knowledge, it would seem best, therefore, to regard the menarche as neither a dependable criterion of maturity nor as necessarily the expression of any narrowly circumscribed stage of sexual maturation. It is, rather, the reflection of a physiologic state which may be achieved at somewhat different levels of maturity by different puberal girls, but which usually precedes by a considerable interval the attainment of the capacity to reproduce.

We have considered the human growth pattern, some limitations of height-weight-age tables based on group averages, the probable relationship of pituitary and gonadal hormones to growth and development, and the value of the menarche as a criterion of sexual maturity.

Because the available time does not permit a more comprehensive presentation, I have limited myself to this sketchy and admittedly incomplete account of some of the physical changes which occur during adolescence. If it has added anything of value to your knowledge of that most interesting creature, the human adolescent, I shall feel justified.

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EXAMINATION OF THE ADOLESCENT FEMALE

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DEVELOPMENT of specialization in medicine has been one of the striking phenomena of the twentieth century. Pediatrics is one of a few specialties that has actually broadened its scope. The pediatrician needs to be an internist in all its major subdivisions. In addition he needs to be something of a dermatologist, something of an allergist, something of an otorhinolaryngologist, something of an orthopedist. He has done a splendid job of convincing the obstetrician that he is best qualified to take care of the newborn and the premature infant, and now I shall attempt to persuade him that he needs to know something more than he may of gynecology.

With the exception of the changes and injuries consequent upon childbirth and with the exception of the gynecologic diseases peculiar to the aged, practically every pathologic condition of the adult female may be found in the adolescent female. Developmental defects, infections and their accompanying discharges, injuries and foreign body problems, tumors, benign and malignant, the endless derangements of the menstrual function, abnormal secondary sex characteristics, these are some of the problems at hand.

Then as the menarche advances to the menacme, disposition, emotions, attitude, and behavior all become the playthings of endocrine readjustment. The reverse process with its accompanying symptoms, as the menacme fades into the menopause, is well known to physicians and laity. The same should be true of this phase of adolescence.

Adolescence in the healthy girl whose home life, school life, and social life are normal requires little or no medical attention. However, even this girl does require understanding parents and teachers.

Let us now consider the duties and responsibility of the pediatrician for the adolescent. Careful examination of the external genitals should be routine after puberty. Accumulations of smegma should be pointed out as a possible source of body odor and local irritation. At this age, the labia majora begin to approximate and eventually completely cover the labia minora, clitoris, and vaginal atrium. There may be evidence of pseudohermaphroditism, such as a protruding clitoris associated with lack of development of the secondary sex characteristics. There may be a bulging imperforate hymen. There may be a total absence of the vaginal atrium and vaginal canal.

The bisexual adolescent should be diagnosed early, if possible, so that an attempt can be made to direct this individual into the proper

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sexual category. Formerly this decision had to be based on surgical exploration. Now endocrines can be identified and gynecography can be done.

An imperforate hymen should be incised, preferably by a crucial incision with the cautery knife, long before the vagina, uterus, and even the uterine tubes become distended with retained menstrual blood. It should be noted that following the release of such an accumulation an occasional death from peritonitis may occur. The girl who has the menstrual molimena without menstrual flow month after month must be investigated for the presence of a maldevelopment which may be anything from an imperforate hymen to a complete failure of union between the Muellerian system and the ectodermal vaginal pouch.

The pediatrician should routinely include a rectoabdominal examination of the genitals in his general examination of the young adolescent. He will thus become familiar with the size and shape of the developing normal uterus and be better able to detect unusual conditions such as a hematometra or hematocolpos. He can learn to recognize a retrodisplaced uterus and even to distinguish between retroversion and retroflexion. Gynecologists know that retrodisplacement occurs in 20 per cent of all women. It is commonly associated with a defective pelvic inclination which is revealed by the near absence of the normal lordosis.

Uterine retrodisplacement is no longer regarded as a condition requiring treatment so long as the uterus is movable. A fixed tender uterus usually means inflammation of the parametrial supports. This is amenable to various forms of heat treatment. The occasional acute retroflexion or acute anteflexion may be responsible for dysmenorrhea. Here the ideal cure is pregnancy. Until then little avails except good hygiene, rest, heat and anodynes.

Variations in the time of onset of menstruation may extend from the eleventh to the seventeenth year without pathologic significance. Irregularity of menstruation during the first year is likewise usually without significance. Anovulatory cycles resulting in prolonged periods of amenorrhea perhaps alternating with menorrhagia or polymenorrhea are not uncommon. No treatment is indicated provided the girl is structurally sound and otherwise well and the blood loss is not excessive.

The appearance of the secondary sex characteristics, i.e., changes in the body contour, development of the breasts, the usual distribution of hair, parallel the establishment of menstruation. These secondary characteristics should be looked for. However, the wide variations in breast development, provided there is any glandular tissue present, need occasion no concern. The hairy pubic escutcheon may become distinctly masculine and be the forerunner of excessive growth of hair on the extremities and breasts. There is no present preventive treatment.

Vaginal discharges may be mucous and noninflammatory or they may be the result of infection. The persistence of a mucous discharge, color-

less, odorless, and without local symptoms, usually requires no local treatment. In my experience these girls are usually hypothyroid and the discharge disappears after appropriate thyroid therapy. Infectious discharges may be gonorrheal or due to any of the usual pathogenic organisms. Very often, these latter appear subsequent to scarlet fever in the adolescent. Now that the sulphonamides are available, these vaginal discharges can usually be cleared up with little or no local treatment. Their persistence in spite of such treatment makes inspection of the vagina and cervix imperative. This can be done readily with either the Kelly cystoscope or the vaginoscope. It is a grave error for the physician to regard the virginal vagina as a *nolle me tangere*. This structure can always be inspected and not infrequently directly palpated without pain and without anesthesia.

The portio vaginalis of the cervix is normally covered by a stratified, squamous epithelium which extends to the external os. This, like the remainder of the vaginal mucosa, is pale pink in color. However, beginning with fetal life, the cuboidal endocervix grows down the canal of the cervix and out onto the portio. Thus at birth 20 per cent of all female infants show a superficial red ectropion encircling the external os. This disappears during the first year. In adolescence this overgrowth tends to recur and may become the site of a chronic inflammation and discharge. It responds readily to 5 per cent silver nitrate applications or to superficial and painless cauterization.

Another source of persistent discharge may be the insertion of foreign bodies into the vagina. Obviously these must be sought for to be found. The cure is self-evident. Rectoabdominal examination routinely carried out may result in the discovery of a tumor, usually of one or both ovaries. These ovarian tumors may be simple cysts, dermoids, teratomas, or occasionally one of the malignant tumors of the ovary. Early recognition of the existence of this pathology in the adolescent is preferable to ignorance of its existence until symptoms appear in the adult or malignancy has advanced to hopelessness.

I have previously indicated that the adolescent is undergoing marked psychic changes as well as physical readjustments. Pediatricians are well aware of this fundamental change just as physicians in general are aware of the psychic changes which occur in menopausal women. A sudden interest in her personal appearance, a sex consciousness which may express itself in exuberance or a new shyness, a slump in scholastic attainments, irritability, crying spells, these are some of the stigmas of this chrysalis stage in the young human female. Pediatricians have learned to help the adolescent through this period with the least possible disturbance, not only by understanding her, but by giving the parents an understanding and by obtaining the cooperation of the girl's teachers. I trust they have learned likewise that it is best to minimize the significance of this passing phase except in these occasional instances in which the services of a psychiatrist are obviously needed.

The same is true of the large range of physical disturbances of the genitals which I have outlined so briefly. They should be recognized early. Thereafter, their significance in general should be minimized. Sooner or later, however, and preferably sooner, if the condition is not controlled, the gynecologist should take hold where the pediatrician leaves off. It has been well argued by those pediatricians who are interested in the problems of the adolescent girl that she is best cared for by the physician she has known all her life. They maintain that this relationship should cease only when adolescence has become maturity. I have no quarrel with this extended assumption of responsibility, provided the young patient gets adequate and competent care.

I am equally convinced that a wider and earlier recognition by pediatricians of the existence of these many gynecologic conditions will redound to the benefit of the young patients whom we once handed over to you in the birth room and whom you now may find require our attention.

MENSTRUAL ABNORMALITIES OF ADOLESCENCE

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Madison, Wis.

FIRST of all I should like to remark that our thought of adolescence is of a process, not of an event. The process is one of becoming adult. It requires a period of years, and it goes on continuously but not always at the same rate of progress. Just where we should date the beginning of adolescence is a subject of discussion. Dr. Wetzel pointed out some time ago that perhaps it ought to begin at about the age of 3½ years, if I remember correctly. Some people think that is a little premature for the normal. It is a question of definitions, but the changes do go on gradually with varying rates of achievement. During the first ten to fourteen years of life we are aware of processes, which are chiefly growth in stature and obviously also in weight, and we see little evidence of maturity in a reproductive capacity until the second decade. Nevertheless, as clinicians we recognize that there are physiologic and also psychologic sex differences long before there are any of these overt manifestations of sex maturity.

Growth must precede the intense maturing process, for we know that when maturity is well advanced, growth ceases and cannot be resumed. In general, that process of growth is limited by union of the epiphyses with the diaphyses of the long bones. The factors which control that ultimate union of the growing parts of the bone are not understood completely. We know that such union and maturity of bones are facilitated by two endocrine factors, thyroid or gonad secretion. I do not believe that these can be held the only factors which control, but that they must be looked on as factors which expedite this maturing process of bone. Statistically speaking, and with rare exceptions, when gonad maturity has arrived, the epiphyses will be found united, and growth will have stopped. That should point our examining attention when we record height, weight, and try to make some comments about the diameters of the chest and pelvis.

Here let me insert a brief remark about the difference between recording the measurements of an obese child and a child with broad pelvis or broad shoulders. When we are talking about these distortions of the growth and developmental process, I think we pay too much attention to obesity and too little attention to the bony diameters involved. I feel sure, in agreement with Dr. Schlutz, that the effect of gonad hormones on obesity is relatively slight. The effect of gonad hormones on skeletal structure is much more definite, and if we will be better observers, we will see such distinctions.

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Growth goes on according to certain definite patterns. We recognize, of course, that there are familial patterns as well as individual patterns. Development goes on according to certain patterns also. It is important, therefore, that we should know something about the family history involved, for then we may not need to be too much concerned about deviations in the development, retardation, or acceleration. The family history is not the only determining factor, but again it needs to be taken into account. A mother who had her menarche at the age of 18 years is not much concerned about her daughter who has primary amenorrhea at the age of 17 years, especially if the mother has had several children, even though she had never flowed until after the age of 18 years. A similar thing might be said of a father who brought to me his two sons with only very slight evidences of adolescence at the ages of 17 and 18 years, respectively, but who remarked that he had had none until he was 18 years of age.

There are several types of hypofunction of the gonads. We need to recognize the different ways in which reduced function of the gonads may be manifested. The normal time of the onset of these physical manifestations of adolescence varies somewhat, as do all so-called physiologic constants. In the young male there is great difficulty in timing the adolescent process. The female has been kinder to the clinician by providing an overt manifestation in the menarche. The time of the occurrence of the menarche in the northern portion of the United States averages about 14 years of age, with a spread from 12 to 15 years. We should consider anything in the period between the ages of 12 and 15 years distinctly within the normal range, 14 years merely being the average finding. The general feeling that southern types mature earlier is erroneous. If there is any deviation, it is a tendency to slightly later maturity, but the deviation is not great. Menstruation occurring for the first time in girls before the age of 12 years can be classified as precocious, and the time interval before the age of 12 years will vary with the gravity of the abnormality. Girls who have not begun to flow until after the age of 15 years may be classified as having a delayed onset of adolescence, and again the duration after 15 years of age will be in proportion to the degree of disturbance. Not all of these patients need treatment, of course. The delay in the onset of menstruation may be an evidence of an endocrine disorder. It may, of course, mean structural anomalies or lack of certain organs, but, granting the careful examination which Dr. Baer has described, so that we know there are genital structures present, the next assumption, if there is no history of pelvic inflammatory disease in a patient with absence of menstruation after the age of 15 years, is a disturbance in the endocrine mechanisms involved.

There are other evidences which mean practically the same thing; namely, underactivity of the ovaries and presumably underactivity of

the anterior pituitary, which stimulates the growth, development, and activity of the ovaries. Those other aspects of hypogonadism and of hypopituitarism have such different forms that they seem to be different diagnostic classifications. Increasingly we doubt that they are different diagnostic classifications. They are merely different aspects of the same type of thing. These different pictures vary from amenorrhea at one extreme to menorrhagia at the other. In between we may find such curiosities as menstruation occurring irregularly, the interval being unpredictable, sometimes short and sometimes long. We may find frequent menstruation, and by that we mean more frequently recurring than once every four weeks, or infrequent menstruation, occurring less frequently than once in four weeks. We may find the duration of the flow very short or long. We may find the intensity of the flow very scanty or extremely copious, and all of those manifestations we now think of as evidences of underactivity of the ovaries, if they are due to any endocrine disturbance whatever.

Now the first impression we worked with from the standpoint of endocrine analysis was that amenorrhea, oligomenorrhea, or infrequent menstruation, represented underfunction. Menorrhagia or frequent menstruation represented overfunction. But in looking for data to substantiate these preconceived ideas, we were forced back to what I announced first, the concept that all these things represent underfunction. Although that may not be the final answer, it is our present conviction. The final answer will have to await very much more exact methods of diagnostic study. Those exact methods which have looked very favorable in the last few years have included biologic assays of blood and urine for the amounts of hormone that were available for study. For the clinician who is not in a frankly investigative clinic with adequate laboratory, I think biologic assays should be put in a pigeonhole and allowed to remain there for somebody else to work out good methods and to interpret them. It is not worth the while of practicing pediatricians to do anything about assays at the present state of development of that method of study.

There are other aspects, of course, of hypogonadism, such as the failure of complete development of the secondary physical characteristics, breast development, development of the adult type of female figure, the hair pattern, and also the psychologic characteristics of the adult as compared with the juvenile female. If these things are delayed in development, one may look for an endocrine background. We are working here in a dangerous field if we make our diagnosis on the basis of these secondary features without having any evidence of underfunction in the primary genital tissues themselves.

If we cannot secure that primary evidence by biopsy assays, how shall we secure it? I think it means tissue studies, and the tissues selected would be the endometrium and the vaginal mucosa. Now obviously one does not want to subject a girl in her teens to the removal

of tissue from the endometrium by biopsy punch or curette, which may require anesthesia and hospitalization in most cases to avoid pain, unless it is genuinely indicated. But if it is genuinely indicated, if there is a reasonable clinical suspicion that one is dealing with hypogonadism and important delay in the adolescent process, then it is well worth while psychologically, socially, and medically to investigate the matter, although obviously not at the age of 14 years. It is much more important to investigate when these changes have not occurred by such ages as 16, 17, and 18 years. This necessitates, of course, the services of a tissue pathologist who knows what the endometrium is like in the normally developed young woman.

There is, however, another tissue which is more readily available, the vaginal mucosa. Although direct inspection of it is not especially helpful, one may study the vaginal mucous membrane cells which are desquamated spontaneously and therefore obtainable by a pipette sample from the posterior fornix or anywhere in the region of the cervix. Such cells may be examined after being smeared on a glass slide and stained appropriately. They will give evidences as to whether we are dealing with estrogenic activity of reasonably normal intensity or with the persistence of the juvenile, immature type. This again requires some experience, but it may be gained relatively easily, and for a gynecologist at least, it is a type of experience which he ought to have as a part of his clinical armamentarium. I doubt that the pediatrician is going to want such experience, because he will not use it frequently. Here is probably a place for cooperation rather than for substitution of the pediatrician's activity for that of the gynecologist.

Dysmenorrhea is a problem already referred to by Dr. Baer, and endocrinologists have hoped that they would have a cure for dysmenorrhea. None of the honest endocrinologists that I know about think they have anything like the answer to the dysmenorrhea problem. I have seen some young women with dysmenorrhea who have been marvelously relieved by endocrine therapy in cases where there was evidence also of delayed development and of infantilism, and where the therapy was directed purely toward facilitating the normal development. I also have seen exactly similar cases that have not been helped one iota. I cannot help feeling, therefore, that we have not gotten to the fundamental method for relieving dysmenorrhea. Probably the reasons are that we do not know the mechanism which causes dysmenorrhea and that there is more than one type.

What can we do in therapy for these patients who have delayed adolescence, incomplete adolescence, or any of these different aspects of hypogonadism? In the first place, I should say we ought not to use estrogenic hormones, ovarian hormones. Those hormones, if given in adequate dosage over long periods of time, will cause development of the uterus, of the vagina, and of the breasts, but they will not stimulate the

development of the ovaries. The most important fundamental thing is that the ovaries of the patient in question should be stimulated and brought up to a normal type of activity if possible in order that as a consequence of ovarian function, the rest of the adult picture may be developed and maintained. If we use large enough doses of estrogens, we can reduce the activity of the anterior pituitary, as a consequence of which we shall further reduce the activity of the ovaries in the patient; therefore, I think in such patients ovarian hormones are contraindicated. On the other hand, we should want to use gonadotropic anterior pituitary materials. The materials that have been available to us in commercial medicine within the last decade are far from satisfactory. Some of them have been inert, some have been very weak, and some have been undependable in their potency. Improvement is being made, so that we are really still in the stage of the earliest and rather unsatisfactory development. However, our patients will not wait. They must be treated with what is available at this time.

In addition to these pituitary gonadotropic preparations, of which there are a few on the market, extracts made from the serum of pregnant mares, of which there are three preparations, have become available more recently. The gonadotropic hormone from the serum of the pregnant mare does stimulate the ovary of the human female and, so far as we know, stimulates it in the same way that the genuine pituitary extract does, with normal function, including pregnancy as a consequence. The material which by all odds has had the greatest publicity and widest use is the chorionic gonadotropin made from the urine of the pregnant woman. This has had its publicity because the commercial firms produced what the men in the laboratories had told them was worth while and came from a relatively cheap source. But it has been produced under a misapprehension, for which the commercial firms were not originally responsible, but which they have been all too glad to continue; namely, that chorionic gonadotropin would stimulate the ovaries of the human female. Apparently the only action of chorionic gonadotropin on the human female is to hold the ovaries in check during pregnancy so that they shall not have a sequence of ovarian cycles maturing ova and forming fresh corpora lutea. This material is apparently what holds the ovary back during pregnancy. It does not stimulate the human female ovary. It does stimulate the ovaries in rats, rabbits, and mice, but I do not think any of us are interested in treating those as patients.

We need to know then what will work on the human female. Curiously, this chorionic gonadotropin, the pregnant women's urine concentrate, does help to bring down cryptorchid testes in many cases, and it has a usefulness there apparently. I think its only usefulness in the human female would be during pregnancy, and by definition of pediatrics, that is where the pediatrician really does relinquish his patients to other physicians.

The other aspect of this problem is the use of thyroid. There is a great deal being said about thyroid. In a gynecologic meeting one is likely to hear it said that thyroid is the only endocrine material worth anything. I am sure that thyroid has been greatly overworked. I think it is overworked badly in the treatment of obesity. I am sure it is overworked in the treatment of other types of disturbances. I know that thyroid is very dependable, but it will not accomplish much unless there is a genuine need for it, and I should like to make a plea for adequate study of the patient before thyroid therapy is prescribed and for genuine evidences of hypothyroidism. These are not easily available by the basal metabolism test before the age of 12 years and are none too certain even in early adolescent years. But there are adjunct tests which are known, one being the study of the bone age. If the bone maturity pattern is not delayed, we have inadequate evidence for saying that there is any significant thyroid hypofunction. The serum cholesterol concentration also is a very useful test. It does not depend on prolonged underfunction of the thyroid but is a cross-sectional test; that is, it tells about thyroid function at the time. If the serum cholesterol concentration is significantly increased above normal levels, we have very good reason to suspect hypothyroidism.

I think we ought to look for such positive objective evidences before we undertake thyroid therapy, not only because thyroid therapy may be otherwise futile, but because it may lead to the use of thyroid to the point of causing mild hyperthyroidism in the patient. Most of our adolescent patients are nervous and jittery enough now and do not need thyroid to add to that feature. Certainly the use of thyroid on an obese child who is not hypothyroid is wasted. It may be worth while having some disciplinary tablets for the child to take along with his diet, but we can use some other placebo with a little greater safety than thyroid.

There are unquestionably cases where adolescent development goes on very early. Precocious adolescence always appeals to me as a sort of tragedy, psychologically and socially. It is a condition to which we ought to be alert. The literature of medicine makes us think that almost all cases of precocious adolescence are due to tumors of the thymus, the ovary, the adrenal, or the pineal. Yet I am convinced that most cases of adolescence appearing well before the age of 12 years are associated with no tumor of any structure, but are due to increased activity of some of the normal mechanisms which bring on adolescence, presumably the anterior pituitary. Those functional cases in which there are no tumor and no pathologic examination post mortem just do not appear in the literature. They ought to in order to give us a more adequate clinical impression for diagnostic studies. Of course, every girl who shows evidences of adolescence beginning at the age of 2, 3, or 4 years, or anywhere up to the age of 10 years ought to be very carefully studied with a search for any possible beginnings of a tumor. If there is a

genuine suspicion I think even a laparotomy might be warranted, for tumor cases ought to be detected early and the tumor extirpated in order to produce a cure and possibly even to save the life of the patient. But I think we must recognize that many of these cases are not caused by tumors.

What can we do in therapy? We can do two things. The first is to explain the matter to the parents, so that there may be intelligent parental guidance of the child who is going to be thrown into the emotional conflict, which we call adolescence, before intelligence and social backgrounds are adequate for that child's problem. Therefore, the parent will need to be more than ordinarily helpful. The same thing may be true of the teachers, and ultimately the child herself must be helped to understand what these things are that are happening in her body. It depends on the individual situation, and here the pediatrician and the parents can cooperate beautifully.

The second, this I admit is theoretical, is to remember what I pointed out at the beginning of my address, the relationship between growth and maturity. If maturity is coming on prematurely in terms of years, we may rest assured that the gain in stature of that child will probably go on more rapidly than the average. She will be tall as well as mature, but she is tall with respect to the other children of her age, and she will ultimately turn out to be a dwarf, because her growth in stature will stop early and she will, therefore, be the short and early matured type. It is possible now to stimulate the growth of dwarf children in many cases by using another type of pituitary material, and we ought to give consideration to growth stimulation in these children if growth is not proceeding rapidly, stimulating that growth before the time comes that the epiphyses unite and the child becomes a dwarfed adult. That I admit is theoretical, but it should be given consideration in the individual case.

Finally, let me suggest one further thing from the standpoint of the endocrine problems of the adolescent girl. In the education of these girls we should think not in terms of giving them one office consultation or one lecture in school about sex hygiene and sex maturity, assuming that sufficient instruction has been given. Adolescence is a continuing process in the girl. There should be a continuing process of education, and I think there is a new point of departure in this education which we medical men and women can contribute. We can point out to girls, to parents, and to teachers alike, that the secondary sex characteristics, both physical and psychologic, are integral parts of the hormone-conditioned development of the person. They are just as important socially and individually as the actual reproductive functions. All of this psychologic picture, not only mate-seeking, but the features which relate to individuality and personality development, are part of the sex maturing process. Sex achieves, therefore, a new dignity and a

new interest. Instead of talking about sublimating the direct sex urge into these other things which are perhaps more socially acceptable at the time, we can point out that what we are doing is not sublimating but emphasizing those things which are important at the time. These include the development of personality traits and of character without paying attention to the physical aspects of sex unless there is some actual physical deviation. I think such a new emphasis on the relationship of these personality traits to the endocrine stimuli gives a new point of approach to sex education which dignifies it. We may treat adolescence as a natural phenomenon for the girl or the boy. This gives us, as clinicians, a very fortunate approach in educating youth.

ENDOCRINE PROBLEMS IN ADOLESCENCE

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TRANSITION from childhood to full physiologic and psychosexual maturity is a particularly trying period for the child, the parent, and the physician. The period of rapid growth, the heightened and modified activities of the glands of internal secretion resulting in the development of secondary sex characteristics, and the accompanying psychologic adjustments to the altered status present to the adolescent a bewildering array of new and powerful forces. The parents are so far removed in age from their own adolescence and are often so strongly influenced by a set pattern of psychic and physical behavior to which they expect their child to conform, that they frequently serve only to intensify the difficulty. The physician, under no little pressure from popular expositions in highly dramatized form of the progress in this field of medicine, is expected to set right the supposed errors of nature by the magic of modern endocrinology.

As regards the physician, the adolescent unhappily falls between two stools. For a short period of his adolescent transition, he still remains the legitimate concern of the pediatrician, and then, by an arbitrary convention, passes into the care of the physician whose training and experience are derived chiefly from the medical problems of adult life. This illogical division of responsibility during such a critical period, exposing the adolescent to the differences in perspective and mode of thinking of these two medical groups, may contribute in no small measure to his difficulties.

The present discussion of endocrine problems of adolescence is based on a synthesis of experience derived from the study of endocrine problems of adult life and a perspective gained from a close cooperation with the Adolescent Clinic of the Department of Pediatrics of the New York Hospital, under Dr. Harry Gordon. From this synthesis, a point of view has been developed which has proved helpful in dealing with these problems. Its thesis is that the majority of the phenomena of adolescence which are commonly regarded as endocrine abnormalities are physiologic variations in the process of maturation occurring in different constitutional types; and that the chief aim of therapy should be to prevent extreme deviations within a given type and, by sane control of environmental factors, to permit the optimum development of the potentialities of the individual. This viewpoint is opposed to the focussing of attention on symptoms and to the growing tendency toward undue interference with the growth and reproductive processes by the use of the many potent endocrine principles now becoming available

From the Russell Sage Institute of Pathology in affiliation with the New York Hospital, and the Department of Medicine, Cornell University Medical College.

for clinical use. The careful application of these principles to adolescent problems of this character will leave behind a relatively small residue of significant disturbances in the physiology of the ductless glands, for which specific therapeutic measures should be instituted. These measures and their effectiveness will be discussed from the standpoint of current knowledge and the writer's experience.

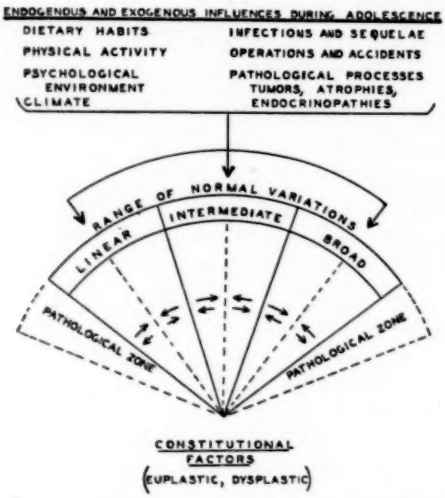

Fig. 1.—Diagrammatic representation of factors operative during adolescence.

GENERAL CONSIDERATIONS

It is helpful to consider adolescence as a prolonged process generally requiring more than a decade for its completion, rather than to emphasize unduly the more striking phase immediately attendant on the onset of sexual function. If one bears in mind that full sexual maturity is not obtained before 17 to 20 years of age, and that somatic development is generally not completed before the twenty-fifth year, one can view with a better sense of proportion the many isolated and transitory phenomena which occur from time to time in the process of maturation. The developmental process during adolescence may be represented as a fan, the configurations of which are usually not clearly seen in childhood (Fig. 1). During adolescence the leaves progressively unfold until the design is completely revealed as the adult state is reached. Segments of this fan are distributed between the three major constitutional types, the linear, the intermediate, and the broad. Passing through the center of each are is a dotted line representing the optimum for each type. The solid lines represent possible deviations within the type. The types are set by inheritance and may either be euplastic (symmetrical) or dysplastic (asymmetrical). The major factors which influence the

extent of the deviations within each type are indicated in Fig. 1. Among these are dietary habits, physical activity, the psychologic environment and the reaction of the individual to it, climate, infectious diseases and their sequelae, operations and accidents, and pathologic processes such as tumors, atrophies, and the various endocrinopathies.

Each type tends to have a fairly characteristic pattern and tempo of development. These patterns can in turn be influenced in their regularity of unfolding and speed of development by the variety of influences mentioned above. Those who deal largely with adults have grown to recognize these types as legitimate and normal variants in the general population and have learned to distinguish, to some extent, the behavior patterns, both physiologic and psychologic, common to the different groups. For whatever reason, we are less accustomed to apply this point of view to the growing child, possibly because it is often difficult to recognize very early the pattern to which the adolescent will eventually conform.

These inherited constitutional factors appear to work in part through an inherent glandular pattern as Stockard has so clearly pointed out by his breeding experiments in dogs. This glandular pattern may function smoothly or undergo certain modifications from a variety of influences. These modifying influences when not too extreme usually act by accelerating or retarding the rate of development. An example of this is the sudden acceleration of gonadal development in the so-called Fröhlich type which often follows on a loss of weight resulting from dietary control and systematic exercise. A common consequence of the fact that the period of sexual development is generally so prolonged and that the glandular pattern does not always work itself out smoothly is the frequency of menstrual irregularities occurring during the first few years after the menarche. An appreciation of the fact that full sexual maturity is not attained for some time after the onset of menstruation, and that some degree of variability is the rule in most pubescent girls should dissuade one from attempting specific glandular treatment of these minor irregularities.

Occasionally, however, the effects of the factors to which we have called attention may be so specific and significant as to alter the entire glandular balance and with it the somatic and psychosexual reaction of the whole organism.

Our aim in dealing with the endocrine factors in adolescence should be for the most part to permit the optimum development of the inherited potentialities. This is best achieved by surroundings that are healthy and psychologically sound, by nutrition adequate in growth factors and adapted to the individual, and by suitable kinds of physical activity. The assistance of the parents should be enlisted by giving them the necessary reassurance and a proper appreciation of the factors involved. By far the greater number of adolescents who are commonly regarded

as presenting endocrine problems will, if properly handled along these lines, develop into effective normal adults.

The remainder will fall into two main groups. The first presents temporary deviations from the normal in an organism which is otherwise capable of effective activity. These are wholly or partially correctable by fairly specific measures. Examples of this group are adolescent goiter, Graves' disease, and maldescent of the testis. The second and numerically smaller group comprises deviations so extreme as to indicate either a developmental anomaly or destructive process or tumor involving a gland of internal secretion. These deviations would extend into the pathologic zone of our diagram.

SPECIFIC PROBLEMS

1. Growth and Nutrition.—By and large, the endocrine products which are now available are of little help in dealing with problems of growth and nutrition in the adolescent. The chief influences in determining stature and form are constitutional and environmental, of which the former is regarded the more important. It is hardly likely that the child whose parents and grandparents are of small stature can be expected to exceed by much the limits of his probable inheritance. Proper nutritional conditions and intelligent attention to his physical development should enable him to make the most of his potentialities. The importance of these factors is seen in the increasing height and weight of successive generations of American college students, to which improved nutritional conditions have undoubtedly contributed. Parents should be urged to cease stressing the significance of size in order to make the child less self-conscious, and to encourage some type of behavior, either in the form of an intellectual pursuit or some physical activity, in which the child gives evidence of special proficiency. The sense of confidence which may be gained in this way is an important aid in the adjustment of the child to his stature.

These observations apply to youngsters who, except for being undersized, are normal in all other respects. A somewhat more favorable though guarded prognosis can be entertained for a small group of cases whose failure to attain normal stature is associated with certain glandular or developmental abnormalities. Evidence is accumulating that some boys with retarded growth associated with hypogonadism gain in height and weight following prolonged treatment with testosterone (10 to 25 mg. two to three times weekly). In many but not all cases, the hypogonadism is associated with a delay in skeletal development. Uncertainty still exists as to whether those given this therapy in their early teens would not have matured sexually and physically had therapy been withheld. In young girls a comparable situation is occasionally met with. Primary amenorrhea is frequently associated with short stature and retarded skeletal development, as well as failure of develop-

ment of the secondary sex structures. Prolonged treatment with estrogens in amount sufficient to develop secondary sexual characteristics will occasionally result in significant increases in height and weight. Apparently the conventional view that the slowing down of the growth process at puberty is due to the increased elaboration of the sex hormones is an oversimplification of a very complex reaction, since it is possible to initiate further growth in these patients by the use of these hormones. Favorable hormonal effects on growth can also be expected from the administration of desiccated thyroid substance when the growth defect is due to hypothyroidism. This problem will be discussed in more detail below. The growth factor of the anterior pituitary gland has been uniformly ineffective in my hands in a variety of conditions associated with retarded growth and development.

With respect to both excessive obesity and undernutrition, a nonspecific approach is, for most cases, the only one available at present. Here, too, the same constitutional and environmental factors operate. There is also an increasing appreciation of the importance of psychologic factors in many cases of obesity, the overindulgence in food serving as a substitutional gratification for more deeply seated discontents and frustrations. An evaluation of the familial background will often afford some idea as to what may be accomplished. The young obese adolescent who usually conforms to the so-called "hypopituitary" type should be dealt with early and watched carefully all throughout adolescence. He should be taught to eat in moderation but to include in his relatively low calory diet enough protein, an abundance of vitamins, and sufficient calcium. No sudden loss of weight should be sought, but rather a slow steady one which will bring the patient to a fairly normal weight consistent with his predicted frame and body type at about 16 to 18. Thyroid extract may be given whenever any evidences of thyroid insufficiency are detected. This is rather an infrequent occurrence, and a relatively small dose of thyroid substance should be sufficient to correct a genuine insufficiency. Except in the presence of hypothyroidism, it is extremely doubtful whether thyroid substance is of any help in reducing weight unless given in amounts large enough to have undesirable effects. Physical exercise is indispensable, but the regime should be carefully planned with the individual in mind. During the onset of puberty, games of endurance should be avoided, as well as sports which stress the competitive element, for they may expose the ungainly youngster to ridicule and hence to a reluctance to participate in athletics. Mass drills are good, and organized games have advantages, apart from the exercise, in terms of social behavior. Best of all, where the expense can be afforded, are individual exercises under expert supervision.

For the other extreme, the thin asthenic adolescent, there is likewise no specific glandular therapy. One should search carefully for psychic factors which may be responsible for the underdevelopment. It is

amazing how many young girls have voluntarily adopted eating habits based on the thin flat-chested, boyish ideal of feminine beauty. These cases are not infrequently superimposed on a profoundly psychoneurotic background of a complex character. Many instances of so-called Simmond's disease appear to be cases of anorexia nervosa and present the necessity for careful psychiatric handling. Frequent feedings of smaller amounts are often beneficial for those who feel uncomfortable after an ordinary meal. Vitamin B complex can be added to the diet with benefit, as well as other vitamins which a capricious appetite may have restricted in the diet. The use of small doses of insulin before meals may occasionally improve appetite and intake, and thus assist in increasing the capacity of the stomach. The influence of exercise in building muscle in this type should not be overlooked. States of undernutrition in young girls are very frequently associated with amenorrhea. When the undernutrition is extreme or prolonged, the resultant depression of ovarian function may be so profound as to lead to a permanent amenorrhea. Hence it is important to check it as promptly as possible. How this may be recognized and dealt with is discussed in the following section.

2. *Sexual Development.*—In young girls, the onset of the menarche may take place at any time from the tenth to the seventeenth year, the average being from 13 to 14 years, according to studies of American girls. The age of onset seems to be influenced, to some extent, by climate, race, heredity, diet, and other factors. Some variability of the menstrual flow, both in regularity and in character, is common in most girls for several years after the menarche. This fact should encourage a conservative attitude toward many of the menstrual problems in this age group. Factors which can be controlled are sudden and excessive loss or gain of weight, neither of which is favorable to normal and regular menstruation.

Amenorrhea during adolescence is of variable significance. It is customary to divide the amenorrheas into two groups. If menstruation has never occurred, they are classified as "primary." If amenorrhea has interrupted a period of cyclic menstrual activity, it is considered to be of the "secondary" type. By the use of the vaginal smear method, this classification can be supplemented by a functional one based on the degree of ovarian underfunction as well as the presence or absence of cyclic ovarian activity. On the basis of the vaginal smear picture, the amenorrheas may be subdivided into three groups. The first is characterized by the constant presence of the atrophic smear indicative of the virtual absence of estrogenic hormone production (Fig. 2). This group includes most primary and many secondary amenorrheas. The second group exhibits smears indicative of subnormal and uniform, i.e., acylic, ovarian activity (Fig. 3). Into this group fall most of the secondary amenorrheas. The third and smallest group shows irregular

cyclic vaginal smear changes, imitating those seen during the normal menstrual cycle and reflecting periodic ovarian activity, which is, however, not sufficient to produce overt menstruation. This picture is seen chiefly in secondary amenorrhea and in adolescents with delayed puberty.

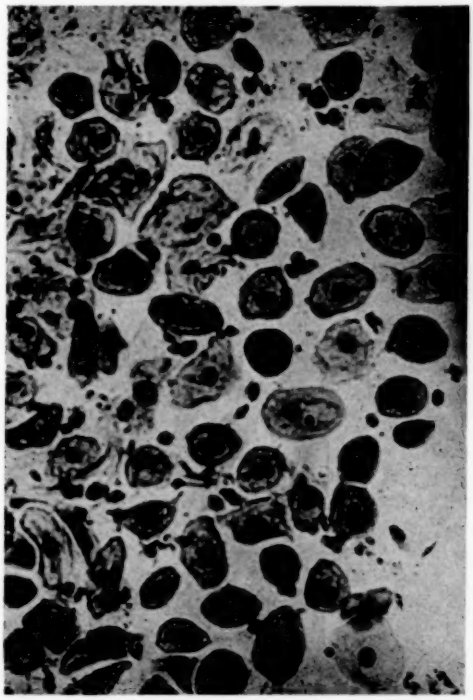

Fig. 2.—An atrophic vaginal smear made up of leucocytes and round or oval cells, with large nuclei, derived from the deeper layers of the vaginal epithelium. This smear picture signifies the absence of estrogenic activity. It is characteristic of the primary amenorrheas, and, when it occurs in secondary amenorrhea, it offers a bad prognosis for the spontaneous resumption of menstruation.

The value of the vaginal smear as a prognostic index and as a guide to the therapy of amenorrhea may be pointed out briefly. In *delayed puberty,* the presence in the vaginal smear of some evidence of ovarian activity with small cyclic fluctuations in association with the development of secondary sex characteristics, warrants a good prognosis for the eventual establishment of normal menstrual function. If, however, the secondary sex structures have not begun to develop by 15 or 16 years of age, and the vaginal smears are persistently of the atrophic type, the likelihood of a true primary amenorrhea is greater than of a simple delayed puberty. *Secondary amenorrhea* is likely to be less significant if the vaginal smears show some degree of ovarian activity than if the smear picture is consistently of the atrophic type. In the former group,

menstruation will often be resumed spontaneously when the basic condition has been corrected by nonspecific measures; whereas, in the atrophic group, a spontaneous termination of the amenorrhea is much less likely. Vaginal smear studies should of course be supplemented by other diagnostic procedures that might throw light on the nature of the amenorrhea. Thyroid insufficiency should be considered and investigated as a possible delaying influence. The excretion of excessive amounts of gonadotropic hormone in some cases of primary amenorrhea and the increased excretion of androgenic substances in the adrenogenital syndrome are of great diagnostic value. Occasionally an expanding sella turcica provides the explanation.

Fig. 3.—An intermediate type of vaginal smear, with leucocytes, bacteria, and partially developed epithelial cells, indication of low-grade ovarian activity. This smear type is the more usual one in the secondary amenorrheas. In the acyclic type, no variations in the smear take place. In the cyclic subfunctional type, fluctuations take place from time to time as the result of periodic increases in ovarian activity, which are, however, insufficient or too brief to lead to uterine bleeding.

The treatment of primary amenorrhea is based on the unlikelihood that either menstrual activity or the development of the secondary sex structures will occur spontaneously. The defect in most cases is believed to be genetic in nature, and to be associated with defective ovarian or pituitary structures, the former occurring more often than the latter. It

is often accompanied by small stature and other developmental anomalies, especially of the bony skeleton. Replacement therapy with estrogenic hormones offers the only means at present of dealing with this condition. The following regime can be instituted with the expectation that the development of secondary sex characteristics will be obtained, that periodic bleeding will occur, and that the adolescent, as a result, will

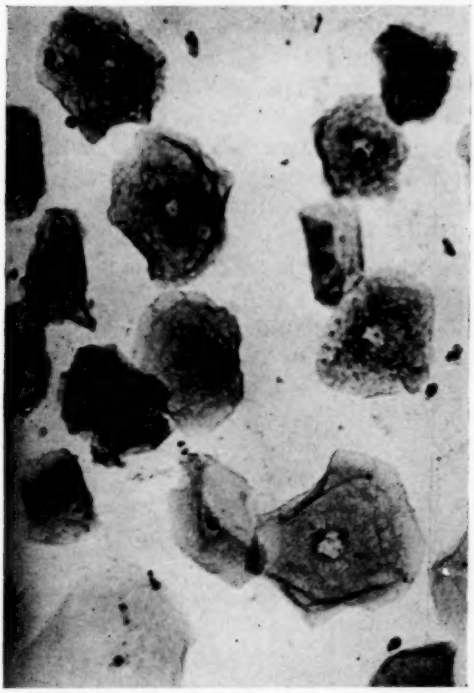

Fig. 4.—An induced follicular smear in a case of primary amenorrhea resulting from complete substitutional therapy with an estrogen. This is similar to the smear picture seen midmenstrually in the normal cycle at the height of follicular activity and represents the goal of substitutional or stimulative therapy in amenorrhea.

be assisted to make a proper psychosexual adjustment to the adult status. Estrogenic hormone is given either intramuscularly or orally during two weeks of each month. That amount of hormone is used which will transform the atrophic vaginal smear characteristic of primary amenorrhea to the cornified follicular type seen midmenstrually during the normal menstrual cycle (Fig. 4). With proper dosage, comparable changes take place in the uterus leading to its enlargement and to the development of a proliferative endometrium. Following the cessation of therapy, pseudomenstruation will occur from the proliferative endometrium, usually about five days after the last dose. Repetition of this therapeutic regime will eventually produce satisfactory development of

secondary sex characters, a more mature and feminine figure, and the enlargement of the infantile uterus in preparation for any pituitary stimulation of the ovaries which might either occur spontaneously or be administered. If vaginal smears are not used as a guide for therapy, the dosage can be worked out empirically although less exactly and less economically. Full substitutional therapy is usually achieved with daily doses of 2,000 to 3,000 R. U. intramuscularly for fourteen days. Here, as well as in the substitutional treatment of secondary amenorrhea, the administration of progesterone after the proliferative endometrium has been built up with estrogen will bring about the formation of a secretory endometrium and a more normal menstrual bleeding. Whether any advantage is gained by this more complete duplication of the uterine changes of the normal cycle is still uncertain. When the oral route is employed, fifteen to twenty times the number of rat units required for injection must be given. After about two years or more of treatment, the newly acquired secondary sex characters tend to persist without further treatment. In some cases, a definite increase in height and weight is also effected. The substitutional character of this therapy and the significance of the bleeding should be made clear to the family and eventually to the patient. What it achieves by assisting the adolescent to make an adequate adjustmer to the adult status can be appreciated only by a comparison with the who in the past have been forced to carry over this defect into adult life. Efforts to stimulate the ovaries of these patients by gonadotropic hormones have been uniformly unsuccessful in my hands.

In cases of secondary amenorrhea of the atrophic or the acyclic subfunctional type in which the amenorrhea has been prolonged for a year or more, the amenorrhea is likely to persist if no hormonal therapeutic measures are taken. This type of amenorrhea is most frequently seen in adolescents who, because of psychologic maladjustments, have instituted a regime of undernutrition. If the correction of the nutritional disturbance by proper dietary habits, with the aid of psychotherapy, does not restore menstruation, a series of treatments with estrogenic hormone, similar to those described for primary amenorrhea, should be instituted. After the third or fourth course, treatment should be interrupted to determine whether a spontaneous rhythm will be resumed. If not, additional treatment with estrogenic hormone should be given, or gonadotropic hormones may be tried. The use of the gonadotropic hormones for the treatment of secondary amenorrheas is still, however, experimental and uncertain. As much as 5,000 to 10,000 I. U. of chorionic gonadotropin (pregnancy urine extract) may have to be given over a period of ten to fourteen days for successful stimulation. By giving small initial doses and then progressively larger ones, these amounts of hormone can be well tolerated by most patients with only minor local or systemic

reactions. A combination of chorionic gonadotropin and follicle-stimulating hormone from the anterior pituitary gives promise of being helpful, but the dosage of this combination of hormones, as well as that of the gonadotropic hormone from pregnant mare serum, remains to be worked out. The possibility, with all gonadotropic hormones available at present, of inducing abnormal ovarian activity should be borne in mind and should lead to their use only with the greatest of caution. It must be stressed that the amenorrheas described above as requiring specific hormonal therapy are much less common than the benign types, for which either no treatment is required or more general measures are adequate.

The more distressing menstrual disturbances such as dysmenorrhea, menorrhagia, and metrorrhagia unfortunately are not uniformly responsive to specific therapy. Careful search should be made for pelvic or underlying systemic disease, as well as for sources of stress or evidences of neurotic behavior. In general, the best results are obtained by attention to general hygiene, by the institution of a proper dietary and physical regime, and by the relief of stress. Many of these conditions appear to be due to the reaction of the adolescent to the relative ovarian insufficiency which may prevail prior to the establishment of the mature type of menstrual function. The individual sensitivity to ovarian insufficiency appears to be related to the degree of psychologic maladjustment. The well-adjusted child is likely to have fewer functional disturbances.

Where dysmenorrhea is so severe as to be debilitating, an attempt to ameliorate the distress by hormonal means is warranted when other measures fail. The unsatisfactory status of the hormonal treatment of dysmenorrhea is apparent from the number of agents employed and the variety of methods of administering them. Estrogens, progesterone, and testosterone propionate are all reported as affording relief. It is hardly likely that hormones with such different actions should produce these effects through a single mechanism. It is becoming evident that when amelioration is obtained by hormonal agents, it is frequently at the expense of abnormal ovarian and uterine function. This is certainly the case with testosterone propionate. With estrogens as well, the effects may not be physiologic. The relief that is obtained from administering large amounts of estrogen early in the cycle is associated with an interference with the luteal phase and the resultant failure to develop a secretory endometrium. Benefit is also reported from estrogens administered premenstrually and with the onset of the flow. The rationale for the use of progesterone rests on its effect on uterine irritability. It should be remembered in this connection that the menstrual cycles in dysmenorrheic women are generally normal ovulatory ones, and that no difference in uterine irritability exists between women with painful and those with comfortable menstruation. The difference would seem to lie in the

manner of reacting to a physiologic process. That this is conditioned to a large degree by emotional stress is becoming more generally recognized and should encourage the wider use of the psychotherapeutic approach in dealing with this problem.

Menorrhagia and metrorrhagia have been considered fully in another paper in this symposium, with whose conclusions as to the desirability of a conservative approach the writer is in accord. These terms comprise a variety of menstrual disturbances of different and still obscure etiologies. In this group of patients, sudden alterations in weight should be avoided. Very encouraging results, as far as the immediate control of excessive bleeding is concerned, follow the use of male sex hormone (testosterone propionate). This hormone causes a regression of the endometrium and generally obviates the necessity for curettage in severe menorrhagia. It also suppresses the gonadotropic stimulus from the pituitary and delays the following menstrual cycle. Dosage is best controlled by following vaginal smears which change to the atrophic type with adequate amounts of androgenic hormone. Once an atrophic vaginal smear is obtained, that amount of hormone is given which will maintain this smear. Treatment should not be too prolonged lest hirsutism, enlargement of the clitoris, or voice changes be produced. The use of androgens therefore should be regarded as a temporary expedient. The proper use of estrogens to build up a more normal proliferative endometrium followed by progesterone to assist in the secretory development should eventually achieve a more physiological control of the menorrhagias, but as yet a dependable regime has not been realized. Mention should be made, for the sake of completeness, of the use of pregnancy urine extracts. Where hypothyroidism exists, the use of iodine or desiccated thyroid is indicated. Every effort should be made to avoid more drastic measures because of the probability that these functional disturbances will eventually correct themselves.

In boys, the disturbance in sexual development encountered during adolescence is chiefly delayed puberty with hypogonadism, with or without maldescent of the testis. While the young adolescent who is somewhat overweight has been shown to attain puberty somewhat earlier than his thinner contemporary, most instances of hypogonadism encountered in puberty are seen in the overweight group with the constitutional type of obesity which unfortunately has been called the Fröhlich type. The delayed puberty in this group has an excellent prognosis under the conservative treatment discussed above.* Only a small percentage fail to come to sexual development. The onset of puberty can frequently be accelerated, best by a low calorie diet and exercise and occasionally by small amounts of thyroid substance where thyroid insufficiency exists. When the testes are normally descended, the use of pregnancy urine, pituitary gonadotropic extracts, and androgenic hormone is generally

*Cf. section on Growth and Nutrition.

unnecessary, although they seem definitely to accelerate puberty, particularly near the time of its customary inception. A valid indication for their use would be the preoccupation of the adolescent with his failure to attain the sexual development of his contemporaries, or the existence of a frank endocrinopathy. When the former exists, these agents can be employed with caution for the purpose of accelerating puberty, and when so used, offer little possibility of harm. On the whole, the more conservative method of diet and exercise provides a much more useful set of habits for the growing boy.

When there is a failure of the testis to descend into the scrotal sac, the use of the gonadotropic principles in pregnancy urine and from anterior pituitary extracts, as well as of the androgenic hormone, appears to be of value in about 20 to 25 per cent of cases. The exact mechanism by which this descent is brought about by the gonadotropic hormones is still somewhat uncertain. They probably act by increasing the interstitial tissue of the testis, which, in turn, elaborates the male hormonal principles responsible for the growth of the secondary sex structures. The androgenic hormone produces the same growth changes by direct substitution. There is a general agreement that a four to six months' trial of hormonal therapy is warranted before resorting to surgery. The age when this treatment may be administered with safety lies between 10 and 12, although it may be given earlier without danger if excessive doses are avoided. In choosing this age period, the following factors were borne in mind: the uncertainty as to the aftereffects of prematurely induced puberty and the prevailing opinion that undescended testes are not likely to undergo significant degenerative changes until the age of puberty. The usual dosage is from 200 to 500 R. U. of chorionic gonadotropin subcutaneously three times a week. Occasionally, doses up to 1,000 R. U. have been employed. The aim should be to secure descent with as little gonadal hypertrophy as possible. In the successful cases there is a tendency for the testes to undergo descent early in the course of treatment although this is not invariable.

The failures with this type of treatment usually reveal at operation anatomic reasons for the maldescent. The postoperative use of the hormone is helpful in maintaining the position of the testicle in the scrotum. Certain by-effects of this treatment should be mentioned. There is frequently a definite increase in the size of the penis and scrotum, with the development of other masculine characteristics and behavior suggestive of premature puberty. Experience has not been extensive enough to evaluate for the younger age group the effect of such changes on future development.

There remain to be considered those cases in which the hypogonadism is part of an endocrinopathy, usually of pituitary origin. This group includes the tall, slender, eunuchoid type or well as cases of hypogonadism of short stature, frequently associated with delayed skeletal growth.

Cases in both groups will often respond to the gonadotropic hormones by an increase in size of the penis and testes as well as a stimulation of sexual function, and by some degree of somatic masculinization. Changes in the secondary sex characteristics can also be achieved by substitutional therapy with androgenic hormones, but stimulative therapy with gonadotropic hormone is more rational by virtue of its effects on the testis itself. It should therefore be given a thorough trial before resorting to substitutional therapy. Not infrequently changes obtained with gonadotropic hormone persist after treatment is stopped. This is much less likely with substitutional therapy. Spermatogenesis should not be anticipated. In the undersized group an increase in height and weight is occasionally seen. The dosage of gonadotropic hormone necessary to produce stimulation of the testis is much larger than that employed in simple maldescent. As much as 2,000 to 3,000 I. U. three times a week subcutaneously may be required for long periods to bring about significant changes. A limiting factor appears to be the individual potentiality of the sexual organs to undergo hypertrophy. In many cases growth is arrested when the testes and penis are still well below the normal size, even though satisfactory function has been established.

3. The Thyroid Apparatus.—The chief disturbances of the thyroid apparatus during adolescence are simple goiter with or without thyroid insufficiency, hypothyroidism, and Graves' disease.

Except in goiter districts where, prior to the prophylactic use of iodine, it was widespread and related in large measure to lack of iodine, simple goiter occurs in those in whom there is some unusual internal or environmental stress or a more labile thyroid apparatus. This latter is commonly familiar. The adolescent goiter is a simple compensatory hyperplasia which can usually be prevented by an increased intake of iodine at puberty, and involuted by the administration of adequate amounts of iodine. Involution usually takes place spontaneously when the stress of puberty is relieved. A convenient form of iodine is the syrup of hydriodic acid which contains approximately 13 mg. of iodine in each cubic centimeter. One or two cubic centimeters daily for six to eight weeks will accomplish involution in most cases. It is usually unnecessary to administer thyroid substance. Whether or not the gland will diminish in size is dependent on the extent of the previous hypertrophy or hyperplasia. If only hypertrophy has taken place, the involuted gland may shrink to its normal size. If a marked hyperplasia has occurred, the gland may increase in size due to the filling of the enlarged acini with colloid. These colloid goiters are usually physiologically adequate but occasionally become a source of concern for cosmetic reasons or because of tracheal pressure. If for either reason partial removal is contemplated, surgery should be deferred if possible until the end of the adolescent period. Since the development of simple goiter in parts of the country outside of the goiter districts is presump-

tive evidence of an unstable thyroid apparatus, it is important to watch these youngsters from time to time and to encourage the use of iodized salt in their homes. A course of iodine for two months in the fall and spring may be necessary, in addition, to prevent further enlargement. Occasionally enlargements confined to one lobe occur which tend to progress in spite of iodine therapy. These are generally due to fetal adenomas which are best dealt with surgically. They are however often difficult to differentiate from involutional adenomas. Thyroid insufficiency may or may not occur in the course of the development of simple goiter. The associated symptoms of thyroid insufficiency are usually not those seen in adult myxedema. Indeed, increased nervousness, restlessness, and the other general characteristics of an organism under a physiologic handicap are commonly seen.

Hypothyroidism in young children may be masked and difficult to detect. It is suggested when there are delayed growth and retarded mental development. These manifestations are usually exhibited in childhood but may persist into adolescence if not dealt with properly. When hypothyroidism develops during adolescence, its manifestations are often so vague as to make diagnosis difficult. Intelligence is usually unimpaired and the general behavior is, as described above, frequently more suggestive of increased rather than diminished thyroid activity. Of diagnostic value are the levels of the basal metabolism and blood cholesterol, the absence of the usual creatinuria of this age group, and x-ray evidence of epiphyseal dysgenesis or of retarded bone age as indicated by a delay in the laying down of centers of ossification. Unfortunately many of these diagnostic criteria are not pathognomonic since their values overlap into the normal range. Their response to thyroid medication generally permits a differentiation. In true hypothyroidism, the response to thyroid administration can be seen objectively in the rise of the basal metabolic rate, fall in the blood cholesterol values, development of or increase in creatinuria, appearance of new centers of ossification, and increase in stature. Cases of retarded development due to hypothyroidism are among the few which respond well to specific endocrine therapy. Mention has already been made of the good response to desiccated thyroid of menstrual irregularities and delayed puberty when they are due to thyroid insufficiency. The dose of desiccated thyroid required for full replacement generally lies between 2 and 3 grains daily.

When Graves' disease occurs in the adolescent, its manifestations are like those seen in the adult and its diagnosis is usually not difficult. The symptoms of the autonomic nervous system instability are likely to be exaggerated and the heart rate higher than would be anticipated from the basal metabolic rate. Occasional cases of marked instability of the autonomic nervous system are encountered in adolescence which are difficult to distinguish from Graves' disease. The creatine tolerance

test is of great value in making this differentiation; a spontaneous creatinuria and low creatine tolerance, which are restored to normal by the administration of iodine, are characteristic of Graves' disease. Graves' disease is most frequently encountered around the inception of puberty at the time that the thyroid apparatus becomes less stable. Its management in the adolescent should be based on the same principles which guide us in dealing with Graves' disease in the adult. Decision as to the choice of therapeutic procedures is most difficult in those cases which develop just prior to the onset of puberty. There is a widespread feeling that every effort should be made to deal with these cases by non-surgical measures first, in the hope that once this stress is relieved, the process will tend to subside spontaneously. While this occasionally happens, one should guard against too long a delay before employing operative measures. In severe cases this conservatism may lead to permanent harm. The best guide is the therapeutic response of the individual case. If continued administration of iodine (30 to 60 mg. a day) is effective in lowering the basal metabolism and maintaining it at normal levels, in abolishing tachycardia, in permitting a gain of weight, and in lessening the nervousness, a conservative regime is advisable. Important adjuvant measures are the relief of strain associated with competition at school and the careful search for other possible psychologic factors. If these beneficial effects are not obtained, or fail to persist when the child resumes its normal activities, the method of choice is subtotal thyroidectomy.

The operative preparation of the adolescent is usually far more exacting than that for most adults with Graves' disease. The prompt response to iodine which justifies operation after a relatively short preparatory period is not as commonly encountered, and more prolonged iodization is generally required. High calorie diets deriving a large part of their calories from carbohydrates, and high in calcium, with additional vitamin supplements, should be employed. Tepid tubs (temperature 98 to 100° F.) twice daily are important adjuvants to other forms of sedation. Iodine should be given throughout the course of therapy and not reserved for a short preoperative period. The explanation for the so-called iodine escape must be looked for in the increased activity of the disease process rather than any "fastness" to the action of iodine per se. If the response to this regime is not completely satisfactory, it is much safer to do the subtotal thyroidectomy in two stages rather than at one operation, thus diminishing the likelihood of a thyroid crisis. As in the adult, attention to sources of emotional stress is no less important after operation than before if one hopes to avoid a recurrence of the disease. Such recurrences appear to be no more frequent in this age group than in adults, except when a strong familial predisposition exists. The use of iodine for a year after operation is recommended as a routine procedure. In the presence of a recur-

rence, it should be given until the process subsides, or, failing this, a second operation is performed.

4. Diabetes Mellitus.—The management of diabetes mellitus during adolescence is based on a consideration of the characteristics of the disease during this period and of the psychologic problems presented by the adolescent in relation to the chronic restrictions imposed on him by his illness. Diabetes, as a rule, tends to increase in severity during adolescence, very likely as a consequence of the accelerated growth and developmental processes involving a complex variety of glandular adjustments. Its course is subject to marked fluctuations, with rapid transitions from marked hyperglycemia and glycosuria to hypoglycemic reactions. On the psychologic and emotional level, the adolescent is more likely than the younger child to rebel against restrictions which he might interpret as impinging on his rapidly developing sense of individuality. Acutely sensitive to the opinion of those of his own age, he is resentful of any enforced behavior which might set him apart from his group and thus contribute to his insecurity.

Fortunately, the present method of treatment is peculiarly well adapted to deal with both the physiologic and psychologic problems of adolescent diabetes. Its essentials are a liberal diet, the use of protamine insulin, and a lessened concern over glycosuria in the absence of ketosis.

The *diet* should be well balanced and provide adequate calories for the growing child, as well as his mineral and vitamin requirements. The caloric needs vary from 35 to 50 calories per kilogram, depending on the age and individual requirements. One and one-half to two grams of protein per kilogram amply meet the needs of the growing child for this constituent. The usual allowance of carbohydrates is approximately 200 Gm., fat providing the remaining calories. Such a diet permits considerable latitude in the choice of foods and attention to personal preferences, and avoids the necessity for the preparation of special meals. With training, the proper amounts of the different food constituents can be selected from the meals served to the rest of the family. The food intake is divided into three regular meals, with small extra feedings at 10:30 in the morning and about 3:30 in the afternoon. A tendency toward hypoglycemic reactions during the night can be dealt with by small feeding, preferably milk, crackers, and cheese, before retiring. Occasional breaches of the diet should not be regarded too seriously, providing highly concentrated carbohydrate foods are not indulged in.

All diabetic children require *insulin,* and all but a small percentage require both regular and protamine zinc insulin, at least in the early years of adolescence. Toward the latter part of adolescence, with a slowing down of the growth and developmental processes and the tendency toward stabilization of diabetes, the number of cases controllable with protamine zinc insulin alone increases. In most cases, the total require-

ment of regular and protamine insulin can be given at one time, before breakfast. Many workers combine both types of insulin in a single injection. Occasionally, a second injection of regular insulin is required, generally before supper. No set rules for insulin dosage can be given since it is dependent on the severity of the diabetes and its characteristics in the individual case. Enough should be given to fulfill the criteria outlined below.

The *proper management* of adolescent diabetes should provide for (1) the liberal diet previously discussed; (2) a gain in height and weight usual for this period; (3) the maturation of the reproductive functions; (4) freedom from any symptoms of diabetes; and (5) freedom from ketosis. Our views have changed as to the significance of glycosuria in the absence of ketosis. It is now generally believed that persistent glycosuria is compatible with good health and normal development; some disagreement, however, still exists as to the degree of glycosuria which is permissible. It is difficult to set an arbitrary value, as, for example, 10 per cent of the ingested carbohydrate, because of the wide fluctuation in glycosuria encountered in children under even the most carefully controlled conditions of diet and insulin. The several purposes of the glycosuria should be borne in mind and that amount permitted which will accomplish these purposes. It avoids the necessity for the too rigid control of the diet and of more frequent injections of insulin, thus diminishing the difficulties in the management of the adolescent. It prevents hypoglycemic reactions. The awareness of the lessened significance of glycosuria should not lead, however, to the adoption of a careless attitude toward the control of diabetes. Every effort should be made to work out the optimum regime for the individual case.

The importance of *education* is obvious; to impart to the child an unemotional, matter-of-fact attitude toward his disease and at the same time an awareness of the necessity for an adherence to the regular regime is a primary responsibility of the physician. The cooperation of the parents will help in this task. The child must be trained to select his diet under a variety of circumstances and taught to administer the insulin carefully. The undesirability of contaminating the bottle of regular insulin with protamine zinc insulin should be pointed out. The significance of infection, however mild, should be stressed. The child and parents should be taught to test the urine for sugar and acetone and be made aware of the premonitory symptoms and signs of acidosis. In the presence of such emergencies they should be instructed not to reduce the insulin dosage even though they eat less, to drink fluids freely, particularly salted broths, and to communicate at once with their physician. The importance of exercise should be stressed, but they should be made aware of the possibility of hypoglycemic reactions and provide for them. The parents should be given a fair appraisal of the child's outlook so that they may be able to guide wisely his subsequent

development. They should be reassured as to the significance of the increasing severity of the disease during early adolescence, lest they interpret it as indicating that the disease will continue to grow progressively worse. They may, with reason, be told that the condition in all probability will subsequently be stabilized and may even improve somewhat. The patient should be encouraged to look forward to a useful, well-rounded life, to continue his schooling in preparation for the pursuit of a gainful occupation, and to participate in social activities and pleasures. This prognosis with certain reservations appears justified by our experience with adolescent diabetes under the present methods of control.

5. Hirsutism, Virilism, and Miscellaneous Conditions.—Brief mention should be made of those cases of excess hirsutism in the young girl, so frequently the source of much mental anguish. They are probably, for the most part, expressions of intersexuality and have no other significance for the individual who is otherwise normal. For this condition there is unhappily no specific therapy. The most that can be done at present is to avoid any overemphasis of this feature which may lead to self-consciousness later, and to employ cosmetic devices, such as electrolysis, wherever possible. A small group has, in addition, associated glandular disturbances such as amenorrhea and pronounced masculinization, in association with adrenal cortical tumors or arrhenoblastomas. Striking effects, with refeminization, restoration of menstruation, and the loss of the abnormal hirsuties, may follow the removal of these tumors. Borderline cases, however, are difficult to evaluate, and for this reason the diagnosis of adrenal adenoma should be made with caution. Of diagnostic value are the high titers of urinary androgenic steroids of low biologic activity in many cases of the adrenogenital syndrome. Much less common in the male, it usually gives rise to precocious sexual maturity and genital enlargement in association with an accelerated growth and muscular development.

Of unusual interest is a rather recently recognized symptom complex in which both genetic and endocrine factors are involved. It is characterized by unilateral cystic disease of the bones, ipsolateral pigmentation, and precocious puberty with subsequent sterility or testicular atrophy. It is distinguishable from osteitis fibrosa cystica by the absence of the chemical changes associated with hyperparathyroidism. No way is known of influencing this condition.

SUMMARY

The point of view has been advanced that most of the endocrine problems of adolescence are expressions of physiologic variations which can be best handled by a sane program of physical and mental hygiene. An essential part of this program consists in the education of the parent to a proper understanding of the factors involved. Those conditions for

which specific glandular therapy exists have been reviewed in the light of our current knowledge. In a field of research which is shifting so rapidly, the practitioner should adopt a cautious attitude toward the clinical application of as yet undigested experimental material.

In preparing this survey, the chief emphasis has been given to methods with which the writer has had direct experience and to those employed by investigators upon whom he has grown to rely. To these workers, too numerous to cite, grateful appreciation is due. Their papers should be consulted for the details of the diagnostic and therapeutic procedures which could be described only briefly here. The writer has also leaned heavily on the experience and counsel of his associates in the Departments of Medicine and Pediatrics of the New York Hospital. This synthesis of experience derived from sympathetic and qualified workers in both adult medicine and in pediatrics appears to offer the most promise for the development of this important field of investigation.

The point of view expressed in this survey, with its chief reliance on sound physical and mental hygiene rather than endocrine therapy, may seem unduly pessimistic in the light of much that is appearing in the current literature. This position, however, is not a negative but rather a positive one, since it stresses the existence of powerful forces inherent in the organism, which, if properly utilized, can make for a healthy transition from adolescence to adulthood. As regards the specific endocrine problems of adolescence which still remain unsolved, a consideration of the progress of the past ten years should be reassuring as to the future.

THE STANDARD METABOLISM OF ADOLESCENCE

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INTRODUCTION

THERE has been considerable disagreement among authorities on the subject of the basal metabolism during adolescence. DuBois,[1] commenting on his original studies, says, "One of the weakest points in the curve of basal metabolism published by DuBois in 1914-15 was a rise in the slope occurring at the ages of twelve to fourteen years, just at the onset of puberty." Bierring[2] in an excellent monograph published in 1931 has very carefully reviewed the literature on this subject from the original experiment of Magnus-Levy and Falk[3] to his own observations in 1931. He points out that the Aub and DuBois standards from 7 to 18 years, because of the number of boys and the circumstances of the experiments, in all probability are too high. This statement has been repeatedly made by DuBois.[4] Bierring further criticizes the puberty rise in the original DuBois, Magnus-Levy, and Falk data on the ground that it is based on an insufficient number of observations. This problem as to whether or not there is a definite increase in the relative heat production at the time of puberty has been the subject of investigation by many experimenters. Göttehe,[5] Lax and Peténvi,[6] and Kestner and Knipping[7] all confirmed this original observation of DuBois. On the other hand, Boothby and Sandiford,[8] Bailey,[9] and Bierring[2] do not favor these findings. All of these observations are based on group sampling at different age periods. In none of these studies were longitudinal observations made on the same children over long-time periods. The heat production expressed in calories per square meter per hour was at a level below that of the Aub and DuBois standards in all of these series.

Because of this disagreement in the various studies, it was considered advisable to adopt the method of longitudinal study in this problem and to investigate the heat production of a group of normal boys and girls at regular intervals between the ages of 10 and 16 years.

TECHNIQUE

A group of thirteen boys and eight girls living at Graham School were selected for this study. All of these children were in a normal state of development at the beginning of the study and remained in that state, except for minor illnesses, throughout the period of observation. In selecting this group of children, an attempt was made to avoid dis-

From the Barbara Henry Research Laboratory of the Department of Medicine, New York Hospital, and Cornell University Medical College.

TABLE I

TABLE OF INDIVIDUAL DATA

BOYS

CASE	NAME	AGE		HEIGHT (CM.)	WEIGHT (KG.)	TOTAL CALORIES PER 24 HR.	CALORIES PER SQ. M. PER HR.
		YR.	MO.				
10	Donald D.	11	2	145.4	35.5	1475	50
		11	7	149.2	37.8	1283	42.4
		11	9	151.1	38.3	1367	44.5
		12	1	152.4	40.3	1460	46.4
		12	7	157.5	45	1510	44.6
		13		161.6	49.9	1647	45.5
		13	7	167.6	55.2	1808	46.8
		14		170.2	58.4	1985	49.6
		14	6	174	63.8	1958	46.6
		15		173.4	62.3	1834	44.2
		15	7	176.5	69.5	1962	44.4
		16		177.2	70.1	2125	47.9
2	George J.	13	1	149.9	39.4	1456	47
		13	6	154.6	42.4	1587	48.6
		13	6	154.9	41.5	1608	49.6
		13	7	156.2	42	1412	43.3
		13	11	159.4	44.9	1583	46.5
		14	5	163.8	49	1713	47.3
		14	10	167.6	51.1	1757	46.9
		15	5	170.2	57	1798	45.7
		15	11	170.8	57.4	1812	45.5
		16	4	173.7	62.3	1649	39.7
		17	2	172.7	64.1	1897	45.1
18	Norman B.	12	1	143.5	40.2	1331	44
		12	6	145.4	41.1	1239	40.3
		12	7	147.3	42.3	1276	40.6
		12	11	147.3	41.1	1247	40.3
		13	5	148.9	43.6	1271	39.8
		13	10	151.1	45.2	1357	41.3
		14	5	153	51.4	1317	37.6
		14	10	154.3	54.7	1488	41.1
		15	4	159.4	58.2	1557	41.1
		15	10	162.6	60.5	1586	40.5
		16	5	166.7	65.1	1793	43.4
		16	5	166.7	63.2	1769	42.9
9	John R.	10	8	139.7	31.9	1350	50.2
		11	1	142.2	33.7	1207	43.4
		11	7	145.4	37.2	1269	43
		12	1	148.6	36.8	1242	41.7
		12	6	151.1	41.3	1158	36.6
		13	1	155.6	46.1	1396	41
		13	6	160	49	1516	42.7
		14		165.1	54.3	1412	37.2
		14	6	167	57.3	1563	39.9
		15	1	172.1	64.9	1749	41.6
		15	6	175.3	66.5	1734	40.4
8	Allan P.	12	6	142.2	30.5	1113	41.8
		12	11	145.1	32	1286	46.2
		13	5	147.3	33.3	1241	43.5
		13	11	150.2	35.2	1176	39.8
		14	4	151.1	36.9	1365	45.2
		14	11	155.6	39.5	1250	39.5
		15	5	158.1	43.1	1548	46.4
		15	10	163.8	47.3	1574	44.3
		16	4	166.4	49.5	1501	40.9
		16	11	169.9	54.3	1607	41.6

TABLE I—CONT'D

CASE	NAME	AGE		HEIGHT (CM.)	WEIGHT (KG.)	TOTAL CALORIES PER 24 HR.	CALORIES PER SQ. M. PER HR.
		YR.	MO.				
3	Condon M.	11	2	132.1	26.8	1187	49.5
		11	7	134.6	27.7	1179	48.2
		12	1	136.8	29.7	1273	49.6
		12	7	139.7	31.6	1274	47.4
		13		141	33.5	1329	48.1
		13	7	144.8	35.9	1353	46.1
		14	7	150	38.5	1596	52
		15	3	156	42.4	1626	49.8
		15	6	158.5	44	1532	45.3
		16		163.8	47.3	1630	45.9
16	Robert S.	11	1	141	34.6	1515	57.4
		11	5	143.5	35.5	1415	49.1
		12		145.7	40	1471	48.3
		12	6	150.5	39	1392	45
		12	9	150	38.9	1253	40.8
		13	4	151.5	42.6	1360	42.6
		13	7	155	44.6	1378	41.3
		13	11	156.5	45.1	1460	43.5
		14	5	161	50.5	1744	48.1
		14	9	164.5	53.3	1766	47.2
		15	4	168	58.4	1805	45.3
		15	9	168.9	57	1626	41.6
20	Coolidge S.	11	10	147.3	38.6	1300	43
		12	2	152.7	42.2	1389	43.2
		12	8	156.9	49.1	1568	44.7
		13	3	161.3	55.8	1567	41.6
		13	8	162.6	54.7	1543	41.2
		14	2	163.8	58.6	1353	34.8
		14	8	163.8	57.7	1592	41.2
		15	2	164.8	64.3	1709	41.9
		15	8	163.5	62.6	1644	41
		16	2	166.1	66.9	1650	39.5
19	Robert St.	10	6	140.3	35.3	1373	48.5
		10	10	144.2	37.5	1300	44
		11		144.8	38.9	1249	41.6
		11	5	147.3	45	1295	40.3
		11	11	150.5	43.9	1290	39.8
		12	2	151	43.9	1326	40.9
		12	9	155	47.3	1200	35.2
		13		154.6	47.4	1132	33
		13	4	156.9	54.1	1477	40.5
		13	10	161	56.5	1392	36.7
		14	3	163	62.7	1566	39.3
		14	10	169	68.5	1674	39.4
		15	3	174	70.9	1749	39.8
17	Charles T.	11	10	148	37.5	1345	44.8
		12	2	150.5	39.2	1367	44.5
		12	4	151.8	40.8	1408	44.4
		12	8	154.9	41	1569	49.2
		13	2	159.1	46.8	1514	43.5
		13	7	161.3	49.5	1533	42.6
		14	2	165.1	55.2	1684	44.1
		14	7	165.1	55.8	1764	46
		15	1	167.6	60	1673	42
		15	7	168.9	60.2	1767	43.8
		16	2	170.2	62.4	1804	44

TABLE I—CONT'D

CASE	NAME	AGE		HEIGHT (CM.)	WEIGHT (KG.)	TOTAL CALORIES PER 24 HR.	CALORIES PER SQ. M. PER HR.
		YR.	MO.				
11	Theodore P.	12	6	146.7	35.5	1427	48.8
		13		148.9	37.3	1262	42.1
		13	5	152.4	41.6	1463	46.2
		13	11	158.1	44.8	1496	44.2
		14	4	162.6	48.1	1710	48.1
		14	11	167	53	1591	42
15	Robert B.	11	2	142.2	30.4	1246	47.2
		11	7	144.8	30.9	1265	46.2
		12	1	148	31.9	1303	46.4
		12	7	150.8	33.9	1091	37.3
		13		152.4	34.5	1181	39.7
		13	6	155.6	37.3	1313	42.4
1	William G.	11	6	142.9	32.4	1327	48.1
		12		145.4	32.5	1099	39.5
		12	5	148	33.9	1212	41.7

tinct racial types. The majority of the children were at least second or third generation Americans and were mostly of Anglo-Saxon stock. They were almost equally divided among the three main constitutional types.

Graham School is a privately endowed institution in a rural district where children are placed either by a guardian or by the state. They live there throughout the year on what is known as a "family basis." That is, children of various ages are grouped together in a cottage under the control of a housemother and housefather. They attend school and do light manual labor. The diet is widely varied and of excellent quality. In general, these children live a life very comparable to boys and girls in any rural agricultural district.

The metabolism studies on these children were made at approximately four-month intervals between 1934 and 1939. A Benedict-Roth apparatus was used, and all of the observations were made by a single individual. The children remained in bed until the time of the test. They then walked a few yards to the laboratory and again rested for one-half hour. Benedict and Crofts[10] have shown that this amount of activity makes no appreciable increase in the heat production. Two six-minute observations were made and the average taken. If these were in disagreement, the test was repeated until check observations were obtained. The experiment was not performed if there was any evidence of respiratory infection. Pulse rate observations were made immediately before and after the tests. Temperature, weight, and height were taken immediately after the experiment.

The children were kept under observation during the entire test. If excessive movement occurred, the test was repeated. In general, the

respiratory curves obtained were of astonishing regularity. In practically every instance there was a sharp decrease in oxygen consumption during the preliminary training period in 1934. However, once this level was reached, there were very few instances of striking variation between one observation and the next.

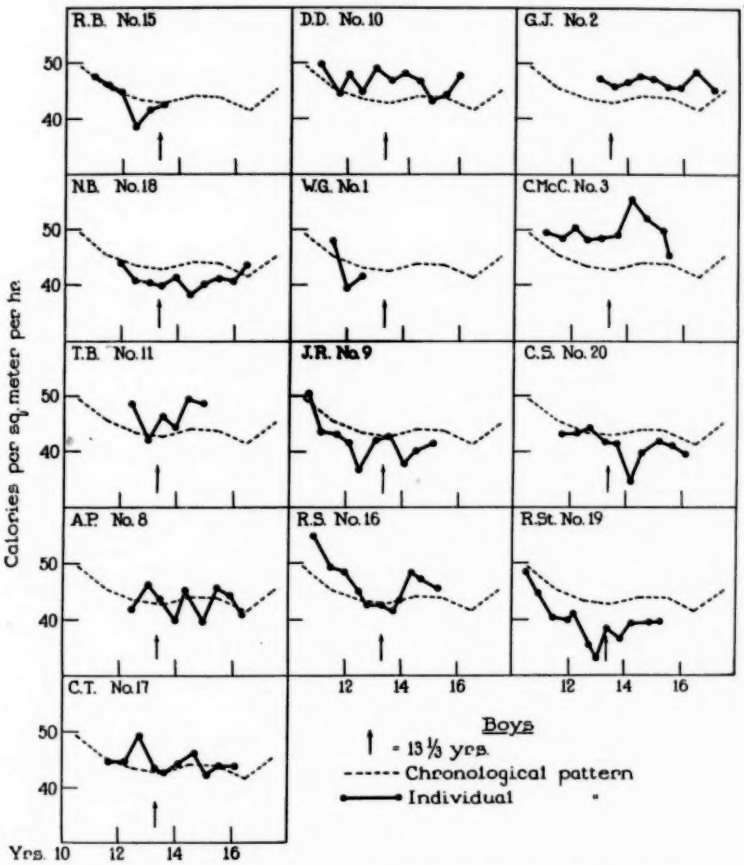

Fig. 1.—Individual data for the boys in relation to the chronologic pattern.

DISCUSSION OF RESULTS

As has been pointed out by many other observers, the training factor is important in some children. In eleven of the twenty-one children studied, the first observations were not appreciably higher than subsequent ones. In eight of the remaining children, a relatively constant level was reached in the second experiment and maintained thereafter. In two instances this level was not reached until the third period of

Table II

Table of Individual Data

Girls

CASE	NAME	AGE		HEIGHT (CM.)	WEIGHT (KG.)	TOTAL CALORIES PER 24 HR.	CALORIES PER SQ. M. PER HR.
		YR.	MO.				
6	Olga A.	10	1	134.6	26.7	1175	48.5
		10	6	137.2	27.5	1147	46.4
		11		141	31.3	1265	47.1
		11	6	144.2	33.5	1164	41.1
		11	11	148.6	35.3	1343	45.5
		12	6	152.4	39.8	1394	44.7
		12	6	152.4	39.8	1340	42.9
		13	1	154	43.7	1508	46.2
		13	6	155	46.5	1421	41.7
		13	11	156	47.7	1318	38.2
		14	7	156	52	1610	45
		14	11	157	52.5	1369	38
21	Grace B.	9	9	134.6	30.1	1187	46.7
		10	1	136.5	31	1187	45.4
		10	7	139.7	31.9	1289	48
		11	1	143.8	34.4	1317	46.1
		11	7	147.3	37.2	1419	47.7
		12	1	151.5	41	1437	45.7
		12	7	155.9	44.9	1463	43.5
		13	1	158.4	48.6	1582	44.9
		13	7	160.3	52.6	1493	40.7
		14	1	162.6	55.9	1631	43
		14	6	163.8	57.7	1536	39.7
4	Irene F.	11	3	145.4	40.5	1302	42.4
		11	8	149.9	46	1433	43.7
		11	10	153.7	47.7	1294	37.7
		12	2	153	49.6	1313	38
		12	9	155.9	52	1287	36
		13	2	157.5	55.5	1438	38.9
		13	7	157.5	57.6	1467	38.9
		14	1	158.8	57.5	1255	33.3
		14	7	158.8	61.3	1297	33.6
		15	2	160.7	60.3	1373	35.3
		15	8	160.7	59.3	1226	31.7
		16	1	160.7	60.8	1399	36
7	Phyllis H.	11	8	133.4	26.3	1186	50
		12	1	135.9	27.9	1098	44.4
		12	7	139.7	29.5	1000	38.6
		13	1	143.5	32.8	1030	37
		13	6	146.7	34.1	1163	40.4
		14	1	149.9	38.5	1121	36.5
		14	6	151.8	38.6	1107	36
		15		153	42.7	1160	35.8
		15	7	153.5	42.4	1191	36.8
		16	2	155.5	44	1203	36.3
		16	6	156	42.9	1176	35.8
5	Edith I.	11	9	140.3	29.4	1056	40.8
		12	1	142.9	30.9	1085	40
		12	4	144.8	26.7	1010	39.3
		12	7	146.1	32.2	1114	39.7
		13	1	149.9	34.3	1011	34.5
		13	7	152.4	37.5	1187	38.9
		14	2	155.9	41.5	1094	33.8
		14	7	158.1	42.5	1251	37.8
		15	1	158.1	44.1	1259	37.5
		15	7	159.7	46.9	1404	40.1

TABLE II—CONT'D

CASE	NAME	AGE		HEIGHT (CM.)	WEIGHT (KG.)	TOTAL CALORIES PER 24 HR.	CALORIES PER SQ. M. PER HR.
		YR.	MO.				
12	Virginia M.	9	11	130.2	26.4	957	40.7
		10	3	132.7	28.1	995	40.6
		10	6	136.2	29.1	1073	42.6
		10	9	136.5	29.8	1027	40
		11	3	140	34.7	1086	39
		11	9	142.2	36.5	1073	37
		12	4	146.1	39.1	1248	41.3
		12	9	149.9	44.5	1341	41.4
		13	3	152.4	48.5	1393	40.9
		13	9	154	50.5	1488	42.5
		14	4	156.2	52.4	1394	38.7
		14	8	156.2	51.1	1393	39.2
14	Lucretia O'C.	11	7	138.4	41.8	1397	46.6
		12	5	144.2	46.5	1259	39.1
		12	11	146.1	54	1482	42.6
		13	4	147.3	54.9	1543	44
		14		148	57.7	1588	44.1
		14	6	149	59.9	1507	41.2
		15		150	62.4	1495	39.9
		15	4	149	58.9	1338	36.7
13	Joyce W.	12	1	149.2	31.9	1140	40.2
		12	6	153	34.8	1225	40.8
		12	8	154.9	35.7	1163	38.2
		13		156.9	37.1	1253	40.2
		13	6	158.1	38.6	1196	37.5
		13	11	160	39.8	1141	35.7
		14	6	160.7	44.8	1100	32.1
		14	11	159.4	42.2	1162	35.1
		15	5	160.7	44.8	1192	34.7
		16		161.3	44.7	1360	39.6
		16	6	161.3	46.1	1286	37
		16	11	161.3	44.7	1316	38.3

observation. Throughout the course of the study an occasional extreme variation occurred during a single day's observation. If any adequate technical reason indicating that this was an error was found, the observation was discarded and the test repeated. If, however, no such reason could be found, the results were retained. In every instance a return to a point near the previous average level, during the next observation, indicated that the previous figure did not represent the individual's true basal metabolism.

In order to determine the relative consistency of the technique, the mean deviation of the individual values from the age pattern was determined. This mean was obtained by dividing the sum of all the positive and negative deviations of each of the different values from the age pattern for the entire group by the number of individual determinations.

The average mean variation for the girls was –0.29 calories per square meter per hour.

In the case of the boys, this analysis gave the results shown in Table IV.

The average deviation for the boys' group was +0.04 calories per square meter per hour.

From this analysis it would seem that the individual observations on each child were consistently in agreement with the general pattern throughout the entire experiment.

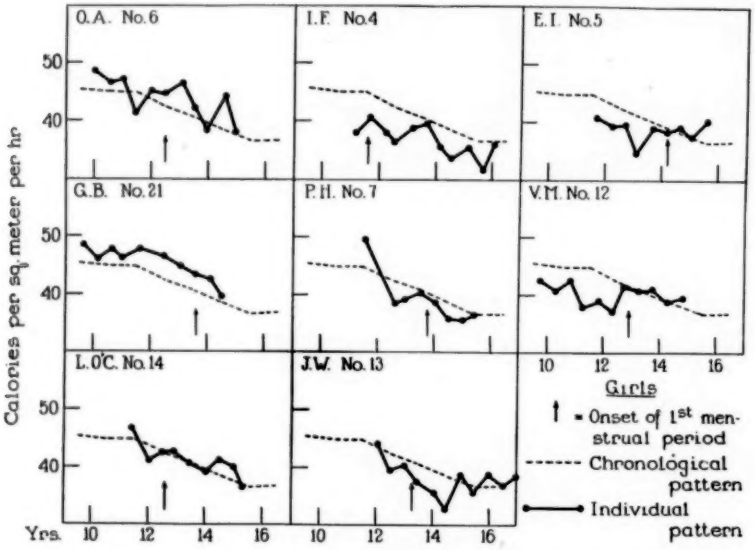

Fig. 2.—Individual data for the girls in relation to the chronologic pattern and the onset of the first menstrual period.

Tables V and VI show the age distribution of the heat production of the boys and girls in our series. As has been shown by all previous observers, the basal metabolism of boys is higher than that of girls. It is of interest that this increase is apparent at the age of 10 years, before marked differences in the skeleton and in the hormone levels of the blood have occurred at puberty. This relative increase is maintained up to the age of 16 years. An attempt has been made to correlate the constitutional type of the individual children with their level of heat production. This did not yield any clue as to the reason for this sex difference. For instance, Case 10, D. D., had a precocious puberty while Case 3, C. M., had marked delay in the development of secondary sexual characteristics. Both had approximately the same levels of basal metabolism. Further analysis of this data and a correlation with skeletal x-rays, sex hormone excretion, and physical changes may yield some clue to this interesting phenomenon.

Reference to Tables V and VI shows a significant drop in heat production between the ages of 11 and 12 years in both the boys and

TABLE III

MEAN VARIATION

GIRLS

CASE	NAME	CALORIES PER SQ. M. PER HR.
6	Olga A.	+1.7
16	Grace B.	+2.8
4	Irene F.	-3.2
7	Phyllis H.	-0.2
5	Edith I.	-1.4
12	Virginia M.	-1.8
14	Lucretia O'C.	+2.3
13	Joyce W.	-1.4

TABLE IV

MEAN VARIATION

BOYS

CASE	NAME	CALORIES PER SQ. M. PER HR.
10	Donald D.	+2.08
2	George J.	+2.58
18	Norman B.	-1.71
9	John R.	-2.61
8	Allan P.	-0.30
3	Condon M.	+4.37
16	Robert S.	+2.45
20	Coolidge S.	-2.34
19	Robert St.	-4.68
17	Charles T.	+0.92
11	Theodore P.	+2.02
15	Robert B.	-0.97
1	William G.	-1.17

TABLE V

AGE DISTRIBUTION OF HEAT PRODUCTION

(CALORIES PER SQ. M. PER HR.)

BOYS

AGE	NO. OF OBSERVATIONS	MEAN
10	3	47.6
11	18	46.2
12	27	43.3
13	30	43
14	21	43.5
15	19	43.4
16	9	42.9

girls. Although this drop is not great enough to stand out in the plotted curves, it is significant when tested by the method of Fisher[11] for determining the significance of the difference between two means. In the case of the boys, $t = 2.2$, and in the girls, $t = 3.4$ calories per square meter per hour. It is hoped that a complete analysis of all the available

TABLE VI

AGE DISTRIBUTION OF HEAT PRODUCTION

(CALORIES PER SQ. M. PER HR.)

GIRLS

AGE	NO. OF OBSERVATIONS	MEAN
9	2	43.7
10	7	44.5
11	13	45.4
12	17	40.7
13	17	40.1
14	16	37.9
15	9	36.5
16	6	37.2

data on these children will yield further information as to why this drop occurs in both boys and girls at that age period. Using the same method of testing, the drop in the mean metabolism at all other age periods between 9 and 16 years was not great enough to be significant.

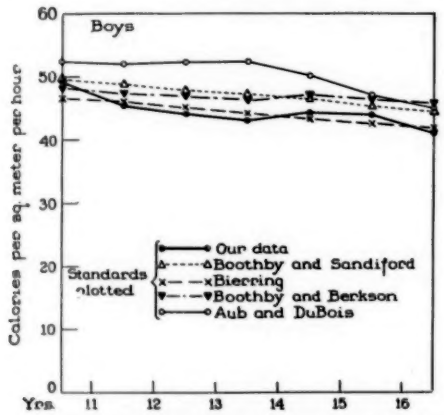

Fig. 3.—Comparison of authors' data with previously published material of other observers (boys).

The values of basal metabolism of boys obtained in the present study agree very closely with those reported by Bierring[2] between the ages of 10 and 16 years. They are in moderately close agreement with the results of Boothby and Berkson[12] and Boothby and Sandiford.[8] They are considerably lower than the original Aub and DuBois standards.

In the case of the girls, our values are almost identical with those of Kestner and Knipping.[7] Our series tends to be slightly lower between the ages of 13½ and 15½ years. In this connection it is of interest that Boothby and Sandiford,[8] Kestner and Knipping,[7] Boothby and Berkson,[12] and our own series are all in very close agreement between the ages of 9 and 11½ years. After that age period the various results diverge.

In general, there was a gradual decrease in the basal metabolism of both the boys and the girls in our study between the ages of 10 and 16 years. Table VII shows the percentage differences between the boys and the girls at the various age levels. The trend of this gradual de-

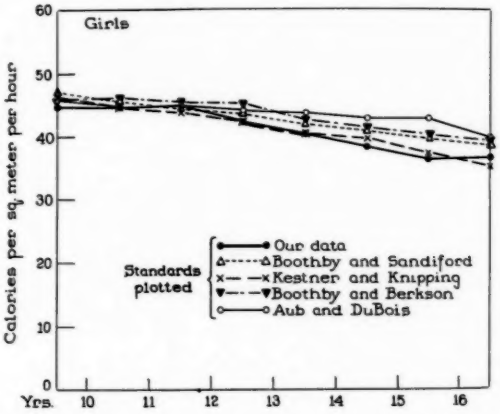

Fig. 4.—Comparison of authors' data with previously published material of other observers (girls).

crease is shown in Fig. 5. This curve of the mean calories per square meter per hour also shows the statistically significant drop between 11 and 12 years of age.

TABLE VII

DIFFERENCES IN MEAN HEAT PRODUCTION BETWEEN BOYS AND GIRLS (AUTHORS' SERIES)

AGE	10 to 11	11 to 12	12 to 13	13 to 14	14 to 15	15 to 16	16 to 17
BOYS	47.6*	46.2	43.2	43	43.5	43.4	42.9
GIRLS	44.5	45.4	40.7	40.1	37.9	36.5	37.2
DIFFERENCE	3.1	0.8	2.5	2.9	5.6	6.9	5.7
PER CENT OF DIFFERENCE	6.5	1.9	5.8	6.8	12.9	15.9	13.3

*Based on only three observations.

Calculation of the data obtained in this study by the various basal metabolism standards in common use reveals information of interesting clinical application. As would be expected, the girls in our series, when calculated by the Kestner and Knipping standards, are all very close to ±0. By the Boothby and Sandiford modification of the DuBois standards and by those of Boothby and Berkson, our mean curves fall in the +0 per cent to −10 per cent range. These same data, calculated by Talbot's standards, show a variation between +9 per cent and −11 per cent.

In the case of the boys, our data, compared to the Bierring standards, show a mean variation between +8.5 per cent and −3.0 per cent.

Throughout most of the curve the variation lies between ±4.5 per cent. These data, calculated by the modified DuBois and the Boothby and Berkson standards, show the average mean to range between −3

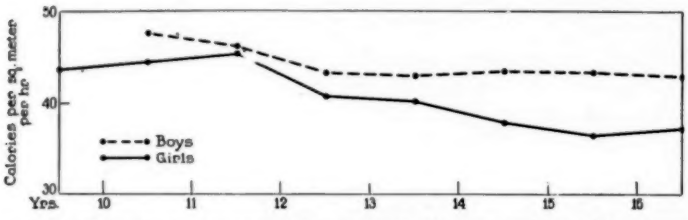

Fig. 5.—The difference in heat production between the boys and the girls at various age periods.

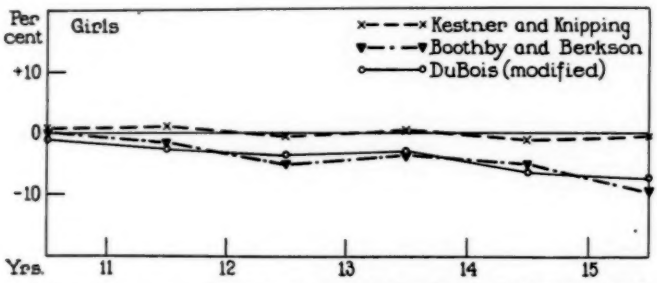

Fig. 6.—Calculation of authors' data by the various basal metabolism standards in common use (girls).

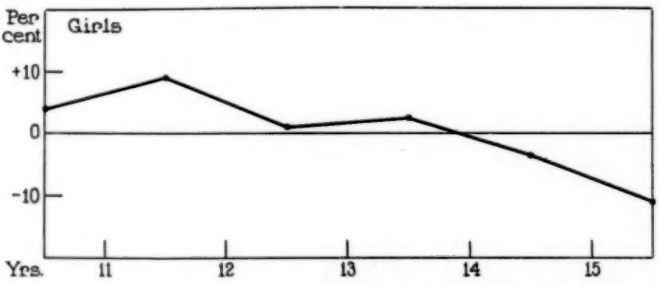

Fig. 7.—Calculation of authors' data by the Talbot Weight Standards (girls).

per cent and −9 per cent, except at the 10½-year period when it is slightly above ±0 per cent. According to the Talbot standards, the mean basal metabolism of the boys varies between +3 per cent and +7 per cent.

From these observations it would appear that for clinical application, the utilization of the Bierring standards for boys and the Kestner and Knipping ones for girls will bring a group of normal children, such as those in the present series, closer to the ±0 per cent figure than any of the other commonly utilized standards.

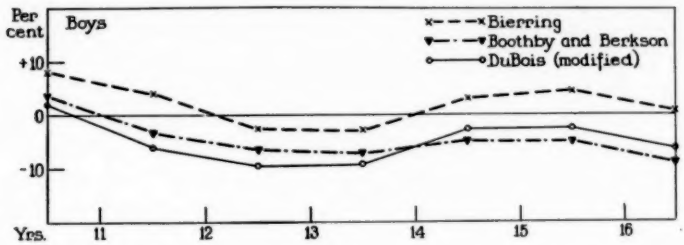

Fig. 8.—Calculation of authors' data by the various basal metabolism standards in common use (boys).

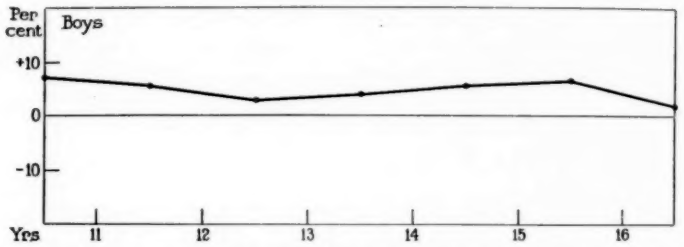

Fig. 9.—Calculation of authors' data by the Talbot Weight Standard (boys).

THE INFLUENCE OF PUBERTY

As has been mentioned earlier, considerable controversy exists in the medical literature as to whether or not an increase in basal metabolism occurs at the time of so-called puberty. This subject has been reviewed by Bierring[2] in 1931 and by DuBois[4] in 1936. Bierring summarized the problem by saying, "We must content ourselves by saying that there is some evidence of a less rapid decrease in the relative heat production at 14 to 15 years, but the experiments are inconclusive, and the number of observations insufficient to warrant a change in the direction of the metabolism curve at these intervals." The use of the term "puberty" in itself leads to confusion in this issue. We now know that secondary sexual characteristics can occur over a wide age period and still be within a normal range. Although most of the authors have used the appearance of these changes as the index of puberty, the range of their occurrence is so wide that, in all probability, if an actual "puberty increase" in heat production did exist, its dispersion would be so great that no significant increase in a plotted curve would exist. It would seem that a better plan would be to attempt to determine whether or not a change in the slope of the metabolism curve does occur at any age period. If such a change is found, then an attempt should be made to correlate it to the multiple changes which take place during early adolescence.

Although most of the observations on the increase of metabolism at puberty have dealt with boys, an occasional study has been made on mixed groups. The data presented in the present study have been analyzed separately for the two sexes.

Reference to Table V or to Fig. 3 fails to show any statistically significant increase in heat production during the period usually referred to as "puberty." The mean values for the metabolism of boys may be summarized as in Table VIII.

TABLE VIII

Age (yr.)	11	12	13	14	15
No. of observations	18	27	30	21	19
Mean value (calories per sq. m. per hr.)	46.2	43.3	43	43.5	43.4

On the chance that the increase in metabolism might be masked by averaging over the yearly age group, a semiannual grouping was adopted.

TABLE IX

Age (yr.)	11½ to 12	12 to 12½	12½ to 13	13 to 13½
No. of observations	9	12	15	14
Mean value (calories per sq. m. per hr.)	44.4	44.4	42.6	42.4

Analysis of this data by the method of Fisher[11] fails to show any statistically significant increase, either in the year or half-year groupings.

The significant drop in basal metabolism occurring in the boys and the girls between 11 and 12 years has been referred to previously. It is possible that this is associated with the intricate mechanism of adolescent growth. In the light of our present endocrine knowledge, a change certainly would seem more likely to occur at that earlier age period than during the 13-year age period at which the suspected "puberty rise" was thought to occur.

Analysis by the same methods of the data obtained on the girls in the present study fails to show any significant evidence of an increase in heat production between the ages of 11 and 14 years. In an attempt to demonstrate a change in heat production in relationship to the onset of the menstrual cycle, the metabolism of the girls was plotted on the basis of the age of onset of menstrual function. This did not demonstrate any significant change in heat production related to the onset of the menses. Similarly, the metabolism of the boys was plotted around an arbitrarily chosen point of 13 years and 4 months. This in no way altered the general downward trend of the basal metabolism during adolescence.

INFLUENCE OF GROWTH

Nylin[13] has studied the relationship of growth and metabolism in boys and girls and believes that periods of lowered basal metabolism coincide

with a minimal increase in height and a maximal increase in weight. Although the problem of the growth rates of the children in the present study will be discussed in another communication, they may be mentioned here in their relation to metabolism.

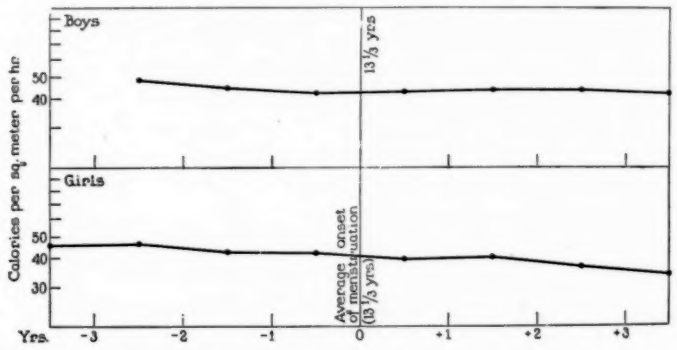

Fig. 10.—Basal metabolism of the girls in relationship to the average age of the onset of menstruation (13 years and 4 months) and the basal metabolism of the boys in relationship to the arbitrarily chosen age of 13 years and 4 months.

The mean growth rate, expressed in centimeters of height, of the boys of the present series was greatest between the ages of 11½ and 15½ years. In the case of the girls, although the growth rate was slower, the rate was relatively constant between the ages of 9½ and 13½ years. After that time period, a slowing in the mean growth rate occurred, while in the boys this did not happen until the age of 15½ years. Thus it would seem that in this series the girls began to stop growing earlier than the boys of a corresponding age.

In the case of both the boys and the girls, the mean of the weight increases paralleled very closely the increase in total height.

The sharp decrease in basal metabolism occurring in both the boys and girls of our series between 11 and 12 years is again demonstrated if the total heat production for twenty-four hours is plotted against increases in total height. In the boys there was a period in their mean growth curve between 138 and 148 cm. when the rate of increase of total calories per day was lessened. This occurred between 11 and 12½ years and corresponds with the decrease in basal metabolism at this age period referred to above. This same decrease is shown in the girls between 132 and 142 cm. This corresponds with the age period 10 to 11½ years when the decrease in basal metabolism was most rapid.

In the boys the increase in the total calories per twenty-four hours paralleled the increase in body weight. In the girls this was true until a mean weight of 52 kg. was reached. This corresponded with a mean age of 14½ years. At this time the rate of increase in total calories be-

came much less marked. This corresponded with a marked diminution of the rate of increase in total height. Although the boys had begun to

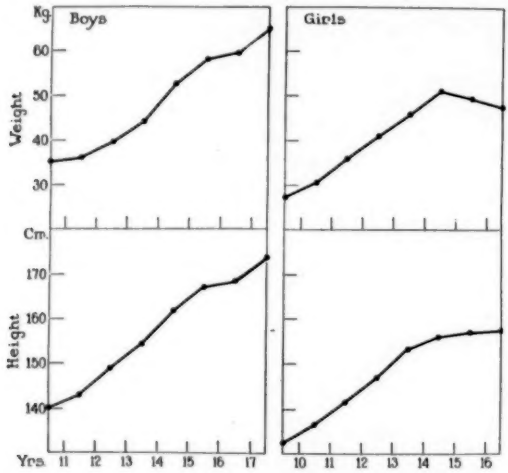

Fig. 11.—The mean weight and height curves of the boys and the girls in relation to chronologic age.

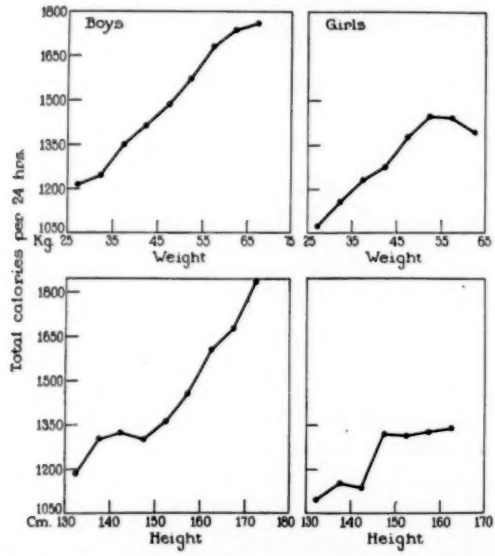

Fig. 12.—The heat production in total calories per day of the boys and the girls in relationship to the mean weight and height curves.

grow less rapidly at the 15- to 16-year age period, their total calories per twenty-four hours continued to increase due to the continued in-

crease in body weight. This doubtless is the "filling out" process often referred to by the laity in connection with this period of adolescence.

SUMMARY

The results of a longitudinal study over a period of five years of the standard metabolism of a group of thirteen boys and eight girls between the ages of 10 and 16 years is described.

The difficulty of obtaining constant determinations with children of that age period did not appear to be a problem. After the second determination, they became accustomed to the procedure and check results were readily obtainable. The average mean variation for the girls was –0.29 calories per square meter per hour, and for the boys it was +0.04 calories per square meter per hour.

In general, there was a gradual decrease in the relative heat production between the ages of 10 and 16 years. This decrease was most marked between the ages of 11 and 12 years on both the boys and the girls. This was found to be great enough to be statistically significant when tested by the method of Fisher.[11]

The boys showed a higher basal metabolism than the girls. This relative increase was present at the age of 10 years, before marked skeletal changes due to sexual differences had taken place.

The values of basal metabolism for boys obtained in the present study agree closely with those previously reported by Bierring.[2] In the case of the girls, they agree almost exactly with those obtained by Kestner and Knipping.[7] The values obtained for both the boys and the girls are considerably lower than the original Aub and DuBois standards.

For general clinical purposes it would appear that the Bierring standards for boys and the Kestner and Knipping ones for girls most nearly meet the requirements for normal children of this age period.

No evidence of an increase in the average relative heat production at the time of so-called "puberty" was found. The onset of menstrual function in the girls was not associated with a change in relative heat production.

The total heat production increases with increasing height and weight. In the girls this increase did not hold true after 14½ years. The total calories of the boys continued to increase after 16 years in spite of a marked decrease in growth rate.

We wish to acknowledge our indebtedness to the Board of Trustees, to the superintendent, Dr. Franklin Thomas, and to the staff and the children of Graham School, Hastings-on-Hudson, N. Y., for their hearty and consistent cooperation through the entire period of this study. We are indebted to Mr. George Bowdery for aid in the statistical analyses.

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OBESITY IN RELATION TO PUBERTY

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A SEPARATE discussion of puberty and adolescence in the obese child is indicated because the discrepancies in the various phases of maturation are even more striking, and the difficulties of adjustment which confront him are even more severe than in the ordinary adolescent. The fears about the normality of his development from which each adolescent may suffer are greatly aggravated in the obese child through the widespread belief that obesity in a child is a signal of sexual abnormality. In recent years there has been published a flood of enthusiastic reports about the remarkable results of glandular injections on the puberal development of obese children. Such reports seem to presuppose that without treatment no development, or only a delayed and abnormal one, would take place. Uncritical and without adequate controls, such reports are misleading. They have added to the fear of possible abnormal development in the obese child. The few and widely scattered statements[1] about spontaneous puberal development do not bear out the gloomy predictions on which so much glandular therapy has been based.

A clue as to the origin of the erroneous belief in puberal delay is given in the diagnostic terminology. Fat children are quite frequently diagnosed as exhibiting the "Fröhlich syndrome." If correctly applied, this diagnosis involves failure to mature sexually. The main features of the case in which Fröhlich, in 1901, first postulated the relationship of adiposity and sexual infantilism to a central lesion may be briefly recapitulated.[2] Fröhlich reported the case of a boy 14 years of age who was slightly undersized and moderately overweight. Signs of sexual development were conspicuous by their absence. Yet this was only an incidental finding. The symptoms that had brought the patient to medical attention were related to a progressing intracranial lesion. During the preceding two years, the same period during which he had grown obese, the boy had been suffering from severe headaches, vomiting, and diminishing eyesight. These symptoms led to the diagnosis of an intracranial tumor, probably in the region of the sella turcica. The symptoms grew progressively worse, so that an operation was decided upon in spite of the great risk and was carried out by von Eiselsberg in 1907. An expanding intrasellar cyst was removed with improvement in the general condition and in the eyesight. The patient continued, however, to be overweight, and he failed to mature sexually.

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The ordinary obese child, except for the existence of adiposity, exhibits none of the dramatic symptoms of these exceptional tumor cases. It has become customary to speak of nontumor patients as "pseudo-Fröhlichs," a device which can result only in further confusion and which serves in no way to clarify the issue. It is not alone the absence of tumor symptoms which differentiates the ordinary obese child from these rare patients with brain tumors. The processes of growth and development, and particularly the puberal development, are fundamentally different.

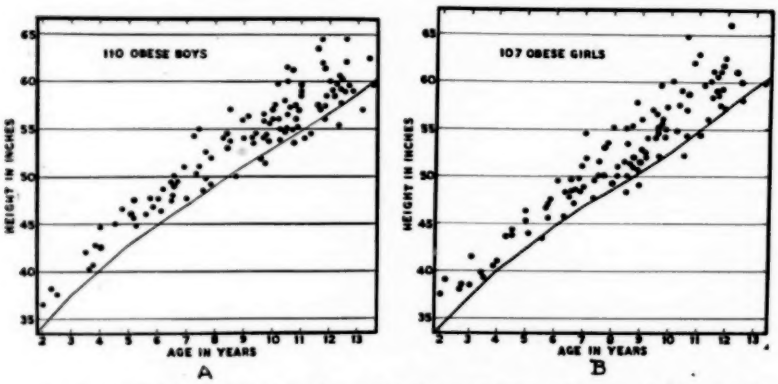

Fig. 1.—*A*, Height of 110 obese boys at first examination. The line of reference is the fiftieth percentile of Burgess' height chart for boys. *B*, Height of 107 obese girls at first examination. The line of reference is the fiftieth percentile of Burgess' height chart for girls.

Puberty represents that phase in the maturation process during which sex functions and characters make their appearance. From this definition it becomes obvious that the diagnosis of failure to mature sexually is highly debatable before the age at which this development may reasonably be expected. The time of onset and the course of puberty stand in a certain relationship to growth and development throughout childhood as has been brought out in numerous studies.

The present report is based on observations made on more than 200 obese children. In all of them obesity had developed before puberty. The weight excess ranged from 25 to 150 per cent. The patients were between 2 and 13 years of age when first seen. They come from different national backgrounds, representing a cross section of the population of New York City. Obesity was the outstanding physical finding. No patient exhibited signs of an intracranial lesion. Many children had been suspected of some endocrine disorder, most frequently specified as hypophyseal. This suspicion could be excluded by the clinical findings and subsequent course.

The heights of 110 obese boys and 107 obese girls are presented in Fig. 1, *A* and *B*, respectively. The normal line of reference is the fiftieth percentile of the growth charts prepared by Burgess.[3] In a

normal group one would expect an even distribution above and below the line. The heights of obese children fall more or less above the line, indicating an acceleration in statural growth. In many instances the height of obese children is several years in advance of the mean. This acceleration in growth is a feature not only of preadolescence when larger variations in body size are common, but it is observed even in very young children. Not only height but many other body measurements are unusually large in obese children.

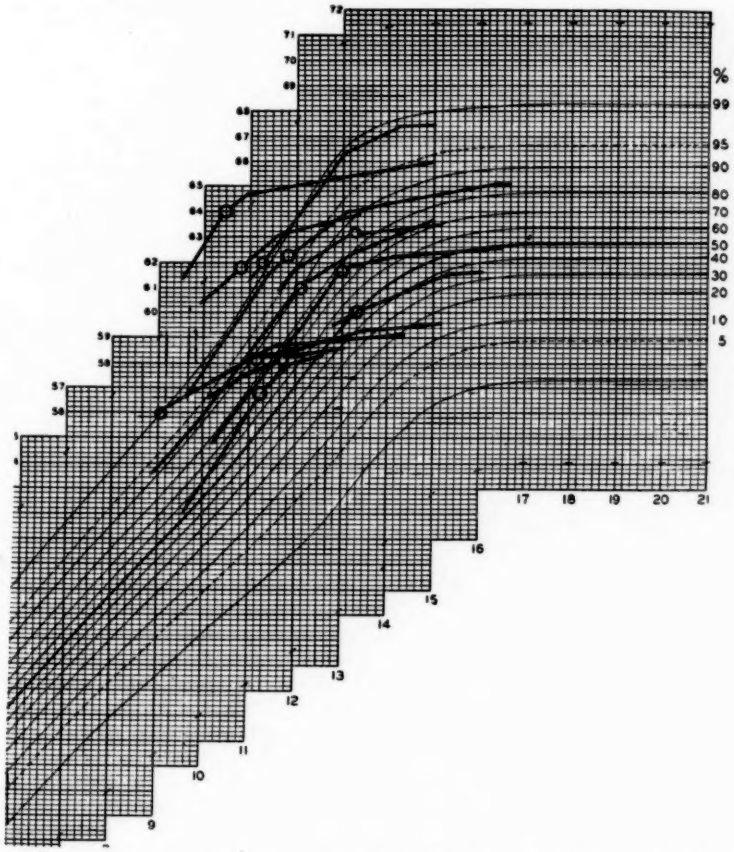

Fig. 2.—Statural growth of twelve obese girls. The open circles indicate first menstruation. The lines are plotted against Burgess' height chart. The figures at the lower margin indicate age in years; those at the upper margin, height in inches. The percentage figures at the right designate the percentiles.

The question of what the final height of these oversized children will be cannot be answered conclusively yet, because the group is too young. In Fig. 2 are presented the growth curves of twelve obese girls who

have been followed for more than one year after onset of menstruation (indicated by a circle). They are shown against the background of Burgess' growth chart. In only one case did growth continue at the previous rate for more than a year. In all other cases a marked change occurred in the intensity of statural growth, and the curves cross their previous percentile lines, falling to a lower level. Some of the overly tall children end up with a stature shorter than average. This behavior is similar to that observed in longitudinal studies of growth: Children with the most advanced growth during preadolescence have a shorter growth period.[4]

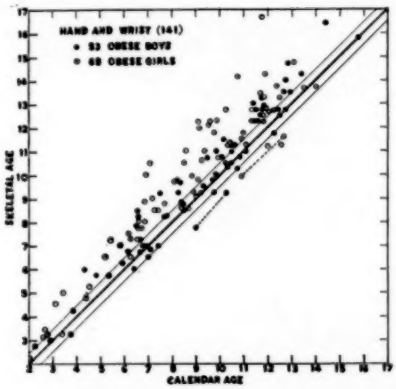

Fig. 3.—Skeletal age of 121 obese children (141 determinations) in relation to chronologic age. The line arising at 45° indicates equality. The two parallel lines allow for an assessment error of ± 6 months.

This pattern of growth suggests acceleration in bone maturation. For a study of the skeletal maturation, 141 roentgenograms of the hands and wrists of fifty-three obese boys and sixty-eight obese girls were rated according to the standards of Todd.[5] The two parallel lines (Fig. 3) allow for an assessment error of ± six months. Forty-five determinations fall within this zone, indicating correspondence between chronologic and skeletal age, or normal bone maturation. Six determinations, relating to four cases, fall below the line. Ninety assessments, that is, two-thirds of the cases, are above the line and indicate advanced skeletal development, sometimes by several years. Other parts of the skeleton were also studied and revealed similar findings. Many obese children with advanced skeletal maturation have been followed, and their early puberal development has borne out the assessment of accelerated development.

Information on the appearance of secondary sex characters and on the menarche of 107 obese girls is summarized in Table I. The recorded findings refer to the age at the time of the last examination. Two children below the age of eight years exhibited the first signs of beginning

puberty. There were only twelve girls above 8 years who failed to show signs of puberal development, and none of these was more than 11 years old. Early signs of puberal development, such as beginning breast formation, nipple changes, and appearance of pubic hair, were present in twenty-five cases. No girl in this group was over 13 years of age. In sixteen girls the secondary sex characters appeared to be fully developed, but they had not yet begun to menstruate. The ages of the twenty-seven girls in the last column refer to the ages at the menarche.

TABLE I

AGE (YR.)	TOTAL NO.	NO SIGNS OF PUBERTY	BEGINNING DEVELOPMENT	ADVANCED DEVELOPMENT	MENARCHE
2 to 7	19	19	—	—	—
7 to 8	10	8	2	—	—
8 to 9	12	9	3	—	—
9 to 10	15	2	8	3	2
10 to 11	13	1	5	3	4
11 to 12	22	—	6	7	9
12 to 13	11	—	1	3	7
13 to 14	4	—	—	—	4
14 to 15	1	—	—	—	1

The age distribution of the menarche of these twenty-seven girls is illustrated in Fig. 4. For comparison, the distribution of the menarcheal age in two groups of New York City children[6] is presented in the same chart. The figures of Boas refer to non-Hebrew girls observed at the Horace Mann School; the observations of Engle and Shelesnyak were made at the Hebrew Orphan Asylum. For both groups the peak of distribution lies between 13 and 13½ years. The highest incidence of menarche in obese girls falls between 11½ and 12 years. Two girls had started to menstruate before 10 years of age. One may conclude from these figures that puberty and menarche appear early in obese girls.

TABLE II

AGE (YR.)	TOTAL NO.	NO SIGNS OF PUBERTY	BEGINNING GROWTH OF GENITALS	PUBIC HAIR	AXILLARY HAIR, ETC.	POST-PUBERTY
Below 9	37	37	—	—	—	—
9 to 10	9	8	1	—	—	—
10 to 11	21	12	5	4	—	—
11 to 12	12	3	3	6	—	—
12 to 13	20	2	3	11	4	—
13 to 14	7	1	1	2	3	—
14 to 15	2	—	—	1	1	—
15 to 17	2	—	—	—	—	2

Concern about possible sexual maldevelopment is expressed more often in relation to obese boys than to girls. In Table II are recorded the progressive phases of puberal development in 110 obese boys at the time of the last examination. No boy below the age of 9 years showed signs

of puberal development. Of the seventy-three boys older than nine years, twenty-six were still undeveloped. No boy above 14 years of age failed to have started his puberal development. As the first phase of puberal development, we have recorded the recognizable growth of the genital organs, which is associated in all cases with a marked increase in the blood supply. This stage was observed in thirteen boys, the oldest of whom was 13 years old. In twenty-four cases the increase in the genital growth had progressed further and had become very marked; in addition, a crop of pubic hair was present. This stage corresponds to the "third phase" in Schonfeld's classification.[7] Schonfeld has made an extensive study of male puberty in New York City. He describes six phases which can be recognized readily by the progressive growth of the gonads and the progressive development of secondary characters. This third phase has been observed in our group in thirty-six patients, twelve of whom since have shown further maturation and are recorded in the last two columns of Table II. These later stages of pubescence are characterized by the appearance of axillary hair, change in voice, male distribution of the pubic hair, moustache and body hair, and the growth of the genitals to adult size. There were two boys above 15 years who were so fully developed that they are recorded as post-puberal.

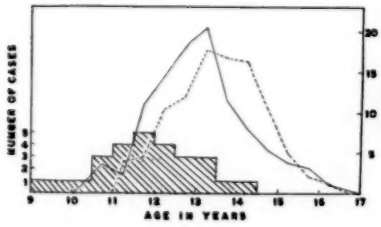

Fig. 4.

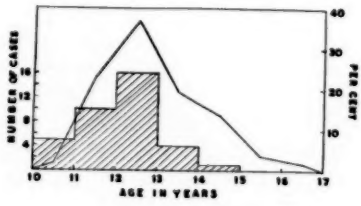

Fig. 5.

Fig. 4.—The area of crosshatching indicates the age of twenty-seven obese girls at menarche. The solid line represents the percentage distribution of the menarcheal age of 226 non-Hebrew girls (Boas); the interrupted line, that of 250 Hebrew girls (Engle and Shelesnyak).

Fig. 5.—The area of crosshatching indicates the age of thirty-six obese boys at the time of puberal development corresponding to Schonfeld's "phase No. 3." The solid line represents the percentage age distribution of fifty-two normal boys observed by Schonfeld.

Since the largest number of observations has been made in the group which corresponds to Schonfeld's third stage, the age distribution of our thirty-six cases is shown in comparison to the normal distribution (Schonfeld's observations) in Fig. 5. The peak of the distribution for both groups, the obese and normal boys, lies between 12 and 13 years of age. There are no indications to support the common belief of sexual retardation in obese boys. One may expect that there are a few obese children who do not reach this stage until after 14 or 15 years; yet they seem to be rare, and they would still fall within the normal range. It is

possible that this late maturing group would recruit itself from those children who do not grow obese until late in childhood or during puberty. In the beginning of this study we intentionally restricted our observations to true childhood obesity. More recently, although we have sought to include cases with late development of obesity, no such case has come to our attention. It would seem that these cases are less frequent than is generally supposed. In a recent report by Werner[8] on delayed

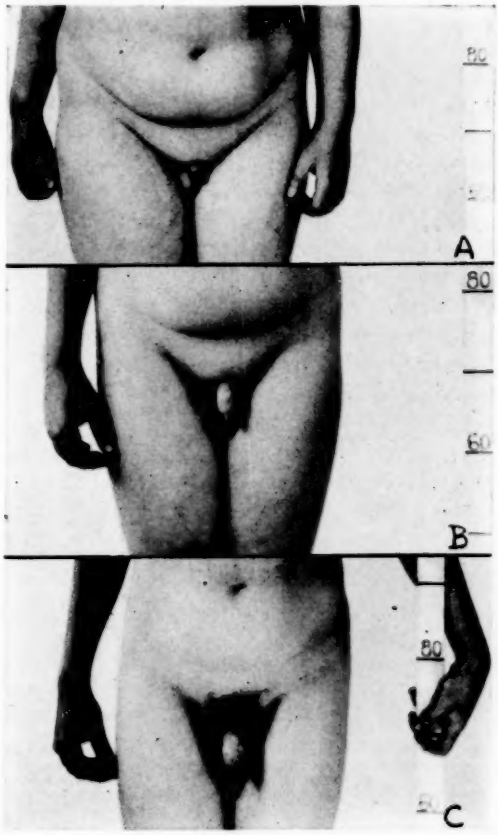

Fig. 6.—Genital development of patient G. T. (*a*) At 11 years and 9 months, no signs of puberty; (*b*) at 12 years and 3 months, beginning growth of genitals; (*c*) at 12 years and 9 months, further increase in genital growth and pubic hair. No glandular therapy.

puberty in obese adolescents, an analysis of the data revealed that only one boy among the eight reported had grown obese after 12 years of age; he was the only one in whom puberal development did not begin until after 14 years of age; he was short and had a spurt in statural growth after 16 years of age. The remaining patients were not dif-

ferent from the group described here. They were advanced in height and had begun normal puberal development between 12 and 14 years of age. In one case bone maturation was markedly advanced.

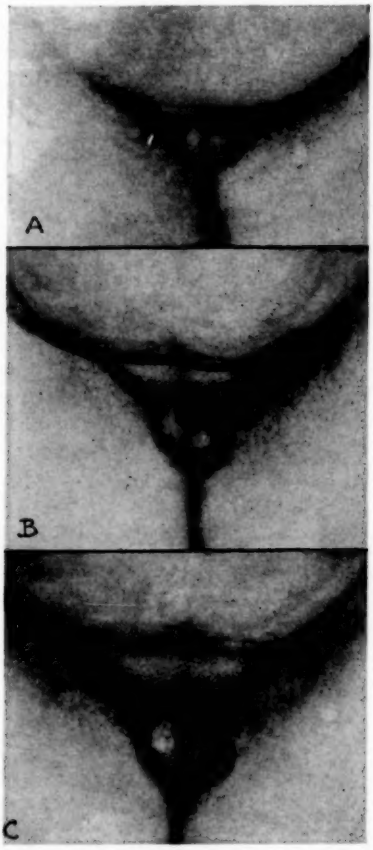

Fig. 7.—Genital development of patient M. P. (*a*) At 11 years and 10 months; no signs of puberal development; weight, 255 pounds. (*b*) At 12 years and 3 months, beginning growth of genitals; weight, 200 pounds. (*c*) At 12 years and 5 months, further increase in genital growth and increased blood supply; weight, 175 pounds. No glandular therapy.

As illustration of the marked and easily recognizable changes which occur in spontaneous puberty in obese boys, I wish to show pictures of some patients who had been suspected of "failure to develop" or "hypogenitalism." The first case (Fig. 6) is that of a boy who was 11 years and 9 months old at the time of the first examination. He was approximately 80 per cent overweight and was very distressed by the fear of "never becoming a real man." Six months later, after dietary regulation and without any glandular therapy, he had lost 40 pounds

in weight, and his genitals showed a definite increase in size and blood supply. There can be no doubt that this represents the beginning of puberal development, and it is borne out by the further progress. At 12 years and 9 months, that is, exactly one year after the first picture was taken, the boy was well advanced in puberty.

Another case is represented in Fig. 7. It shows development in a tremendously obese boy who is still under observation. At 11 years and 10 months the boy weighed 255 pounds and showed no signs of puberal development. Four months later his weight was down to 200 pounds and the scrotum and testes appeared larger. After two more months, when he weighed 175 pounds, the next picture was taken, which clearly shows the progress in sexual development.

It has been my impression that puberty takes a more rapid course in patients of this age period if they lose their excessive fat. Yet obese boys undergo spontaneous puberty even if they remain obese or even gain more weight. It is difficult to evaluate the available data and to prove whether or not weight reduction has a significant influence on the pattern of puberal development, since the normal development shows such a wide range and variation.

Whether or not puberty has a decisive influence on the weight of the children cannot be answered at present. Further follow-up observations on this large group will eventually clarify this problem. There certainly is no automatic transformation of a fat child into a slim and athletic adolescent with the appearance of puberty. Loss of weight and the achievement of normal body proportions seem to be a more complex problem than the result of mere physical sexual maturation.

The presented data reveal that obese children are advanced in statural growth and maturation. Early puberal development is the rule for obese girls and is not uncommon for obese boys. Nothing in this study justifies the assumption that obesity in childhood is in any way associated with hypogenitalism or delayed puberty. Longitudinal studies of growth in large groups of normal children[4] have clarified the relation of puberty to growth during childhood. Tall, rapidly growing children tend to have early puberty and to stop growing at a younger age. The growth pattern of obese children represents an exaggeration of this normal trend.

One might raise the question of how far overnutrition is responsible for this accelerated development. I am not able to state on the basis of my observations whether the increased rate of growth is a cause or only the result of the large food intake. It is possible that the central regulation for both, the intensity of growth and weight regulation, is disturbed in these children. Little work has been done on the effect of long-continued overnutrition. Most studies deal with the result of undernutrition. The large-scale experiment of the World War demonstrated that long-continued undernutrition influences growth and

maturation of otherwise normal children.[9] Long-continued malnutrition during childhood results not alone in deficient weight but is associated also with marked statural retardation and delay in puberal development. It is not unreasonable to suppose that persistent overnutrition has the opposite effect and is, at least in part, responsible for the rapid development of obese children.

The findings are in sharp contrast to all theories which attempt to explain obesity on the basis of hypothyroidism and hypopituitarism. Hypofunction of either of these glands results in retardation of growth and puberal development, a finding which has been firmly established in clinical observation and experimental endocrinology. Since obese children show acceleration rather than retardation of the growth processes, it is not logical to explain the obesity as resulting from deficient function of these glands. The need for endocrine therapy thus loses all factual support. The sweeping claims of the remarkable effects of glandular injections on the puberal development of obese children say actually no more than that puberty takes place, an event which will occur just as impressively if the patients are not subjected to long-continued, and exceedingly expensive, glandular treatment.

This statement, however, does not imply that obese children and adolescents should not be treated. On the contrary, these children need help, not for a fictitious glandular disorder, but for their real difficulties, which interfere with their well-being and adjustment. With increasing age they suffer more and more from their conspicuous and ungainly appearance, and they are under a severe social and psychologic handicap. A reduction of the excessive weight is indicated; yet the patients frequently show marked resistance against any changes in their eating habits, even if they want to lose weight. This paradoxical behavior becomes comprehensible if we understand the development of obesity in a child. Overeating and inactivity are part of a behavior that indicates a disturbance in the maturation of the total personality. In contrast to the accelerated physical and good intellectual development, obese children show a marked retardation in social and emotional maturation. Such a disharmony, which may occur in the development of many normal adolescents, has been present in the obese child throughout his childhood. His behavior has been greatly influenced by his environment, which is characterized by an overprotective and oversolicitous attitude of his parents. It is, therefore, even more difficult for the obese child to gain personal independence and to find a balance between the different phases of his development. In his conflicts, uneasiness and insecurity, he turns to overeating as a source of comfort and enjoyment. He cannot be expected to give up this pleasure until he has found new sources of satisfaction. Treatment of an obese adolescent means, therefore, more than calculating a reducing diet. He needs help for his personal difficulties. Whether or not treatment will

be successful, or whether or not he will "outgrow" his infantile fatness, will depend largely on the adjustment which he is able to make during this critical period of his life.

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WHAT TO DO ABOUT THE FAT CHILD AT PUBERTY

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OBESITY is seldom a problem in early or late childhood. It usually becomes one at or near puberty. Obesity at that time reaches a sort of peak, and on account of its physical, social, and medical aspects, is of concern to both parent and physician.

Obesity at puberty may be of various origins. For practical purposes it often is divided into two principal types: obesity of endogenous origin, and obesity of exogenous origin. In the endogenous type there are the more or less well-defined somatic characteristics and metabolic changes of endocrine dysfunction. This may be thyroid-pituitary, hypothalamic-gonadal, or a combination of several. In the endogenous type, also called simple obesity, it is a question of excess food intake over what is needed for expended energy, with resulting deposit of fat.

It is well recognized that there is a constitutional element and a familial tendency in many cases of obesity, especially of the simple type. Racial factors also enter into the picture. Sex does not play so great a role in children as it does in adults, but there is a preponderance of obese girls over boys at puberty and definitely beyond that period. I would be inclined to agree with the opinion of Wilder, that in most cases of juvenile obesity not referable to gross endocrine dysfunction or neoplasm or acquired cerebral lesion, the constitutional and hereditary element is a big factor. The constitutional tendency and pattern run in families. Von Noorden estimated that 80 per cent of all cases of constitutional obesity were familial.

The mechanism of weight regulation may be hormonal or nervous or both. Whether these factors influence the metabolism directly is still, in large part, conjectural and will probably remain so until distinct abnormalities in the metabolism of obesity are demonstrable. The hormonal influence is, of course, paramount and unquestionable in the well-defined endocrine dysfunction case, whether it be thyrogenic, pituitary, or gonadal, but the factor which is so prominent in simple obesity, big appetites and lack of exercise, also plays a big role in the excessive obesity of the endocrine dysfunction case.

Is the hormonal factor important in juvenile and adolescent obesity, and do endocrine dysfunctions of varying degree really play a big etiologic role? It is a popular conception among the lay public, especially educators, and a large part of our profession that they do. There is a growing conviction among those who have given much study to this problem that hormonal influences play a minor role in the development

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of obesity and have been overemphasized altogether too much. Where there is proved endocrine dysfunction on the basis of aplasia, degeneration, or neoplastic change, causing hormonal inadequacy or deficiency, the etiology is clear. With diminished thyroid activity and concomitant reduced expenditure of energy, somatic changes comparable to obesity will occur. The myxedematous child is not always fat. The weight increase in tissue is largely due to an increase in albuminous fluid. True fat is not much in evidence. Unless other unmistakable evidences of myxedema are present in the case, a metabolism rate not in excess of –20 hardly justifies the diagnosis of thyroid hypofunction, and of thyrogenic origin of the obesity.

The obesity associated with lack of development of the genitals, commonly known as Fröhlich's type, the adiposogenital dystrophy, is believed to be due to hypofunction of the hypophysis. The clinical picture is familiar: The disproportionate distribution of fat with predilection for the pelvic girdle, the thighs, and the upper part of the arms and over the shoulders; the relative thinness of the wrists and ankles and the slender tapering fingers, as compared with the thick wrists, large ankles, and short, squat hands of the child with thyrogenic obesity; the face with its childlike, doll, and peaches-and-cream appearance; the dwarfing or excessive stature which occasionally may be part of the picture. Everyone with considerable clinical experience will agree that the extreme or perfectly typical case of adiposogenital dystrophy or pituitary hypofunction case is rare, but that all grades of transition between it and the more usual forms of generalized obesity do exist and are commonly met with.

Is pituitary hypofunction really the principal determining factor, or are there other etiologic factors not directly concerned with the pituitary in the picture? There is considerable evidence that there are, and that these are largely concerned with the hypothalamic region.

There is little evidence to indicate that secretions of the gonads play more than a minor part in the control of weight or are etiologic factors in juvenile obesity.

Is there some neurologic control or nervous regulation of fat deposit? There is some evidence to support this view. The hypothalamic region and structures near it, such as the tuber cinereum and the walls of the third ventricle, seem involved, and also the forebrain. Lesions of the latter cause tremendous bulimia, with resulting excessive obesity. In cases of adiposogenital dystrophy which have come to autopsy, lesions found in the majority of cases were not in the pituitary body but in the hypothalamus.

If the hypophysis is removed cleanly in the experimental animal, no obesity will follow. If, however, the hypothalamic region is injured, obesity will follow rapidly with or without disturbance of the hypophysis. Lethargic encephalitis is often followed by development of marked

obesity. This disease practically never affects the hypophysis but, by preference, does attack the mesencephalon and diencephalon. The fat deposit may be generalized or may have the adiposogenital dystrophy distribution. The apparent relationship of organic disease of the brain to the development of obesity raises the question as to whether nerve structures in the hypothalamus are not concerned normally in the control of body weight.

May not simple obesity be due in part at least to functional irregularities in these structures? This concept would go far to explain many cases of juvenile obesity in which there are definite adiposogenital dystrophic characteristics with or without excessive food intake.

In contrast to the endogenous type, there is the simple exogenous or alimentary type of juvenile obesity. It originates entirely in environmental factors. The obesity is the result of consumption of food far in excess of the energy requirements of the child. Storage of fat in the form of adipose tissue is the inevitable result. Indolence and lack of exercise are often abetting factors. Significant psychiatric phases, such as parental overprotection, etc., often play a prominent role in the picture. Bruch states that in 80 per cent of her patients, the food intake admittedly exceeded that of nonobese children. In the remaining 20 per cent, the food intake had been excessively high, at least during the period of rapid increase in weight. She also found the amount of energy released through muscular exercise to be smaller than normal in 70 per cent of her cases.

The fundamental cause of obesity of whatever type is an imbalance between energy intake and energy output. In endogenous obesity there is lowered metabolism resulting from disturbed hypothalamic or endocrine function. In the exogenous form, the obesity is the result of a maladjustment between food and exercise. These patients do not have abnormally low basal metabolic rates. All of their metabolic functions, as a matter of fact, are quite normal, and all energy exchange moves within normal limits.

The caloric requirements of obese children are not fundamentally different from those of normal children. The increased urge for food which many of them have may have a physiologic basis or may be psychologic in origin.

There are in the obese child at puberty many psychologic and psychic factors which may play an important role. Among them may be mentioned maternal overprotection and fear in the mind of the parent of sexual maldevelopment in the child. To the mother of the obese child, food often stands for more than caloric value. It represents health and security, and often love and affection. The problem may be in the child himself and express itself as a defense reaction. The inability common in obese children to express themselves by constructive activities

or to find an outlet in satisfying relations may find its appeasement in an increased intake of food.

For convenience of diagnostic appraisement of the obese child at puberty, I would suggest four clinical divisions based largely on somatic characteristics and usual laboratory data, such as basal metabolism, sugar tolerance, and blood pressure readings.

1. True Hypothyroid Obesity.—The limbs and the trunk are obese and the limbs more obese and shorter than would be proportionate even for the obese trunk. The blood pressure is low and the basal metabolism is decidedly below normal, often as much as 40 or 50. The sugar tolerance is generally normal. There is practically always mental retardation of varying degree.

2. Hypothalamic Obesity.—The trunk is obese but the limbs are not. They may be even thinner and longer than would be proportionate for a normal trunk of the patient's size. The blood pressure generally is low. The basal metabolism is within normal limits. The sugar tolerance may be increased. The mentality is usually entirely normal, or may be even superior.

3. Comparatively Rare Form of Frontal Lobe Obesity.—The limbs and trunk are obese, but they are in proportion. The child has generalized obesity, voracious appetite, and commonly shows some degree of mental retardation. There is motor hyperactivity. The blood pressure is normal and the basal metabolism rate and the sugar tolerance are entirely normal.

4. Simple Exogenous, Environmental Obesity.—The child has symmetrical obesity. The family almost always has provided more food than was essential for his activity. The condition is familial in the sense that the whole family probably eats too much. The physical activity of these children is reduced. Their blood pressure may be elevated. The basal metabolism is within normal limits. The sugar tolerance in the course of time becomes elevated.

Clinical observation, aided by the laboratory data enumerated, will differentiate the various types quite readily and will determine the prognosis and indicate the therapeutic approach. The treatment of juvenile obesity depends on the underlying cause.

If it is thyrogenic and one is dealing with the hypothyroid or myxedematous state, the principal remedy is thyroid extract in adequate dosage. Special dietary regime or restriction of diet is not called for. The therapeutic results in general are rather disappointing in this form of obesity. The striking and unpleasantly disfiguring somatic changes will be altered very little, even after prolonged and intensive treatment. The psyche and intelligence will improve. The skin will be less dry, and chronic dry skin eruptions will improve or disappear. The general tissue tone will return more to normal as will also the low metabolic rate. Excessive weight in this type is reduced only with great difficulty and is quite uninfluenced by dietary restrictions.

In the hypothalamic form, the situation is somewhat different. In the rare case of supracellar cyst or hypothalamic neoplasm, successful operative procedure only will cause improvement. Deep, intensive irradiation may cause marked improvement and should be tried. Sharp dietary restrictions, combined with the use of thyroid extract, may give temporary relief. Pituitary preparations are uniformly ineffective in this condition. Effects such as are seen in the experimental animal are not seen in man.

In the much more common classical hypothalamic form without tumor, the results of treatment are better unless the weight increase has really assumed formidable proportions. I have seen girls and occasionally boys weighing in excess of 250 to 300 pounds. The average hypothalamic case of obesity will respond quite well to a reducing diet combined with some endocrine therapy, notably thyroid and occasionally gonad, where there is genital infantilism. With full establishment of puberty, the process may become arrested completely and the excessive gain stopped. A planned activity program should accompany the dietary regime.

In the grossly obese hypothalamic case, the management is much more difficult and the results not so promising. With excessive reduction, these children often develop alarming symptoms, such as frequent attacks of syncope, great weakness, hypoglycemic states, and marked anorexia. The patient should not be ambulant during active treatment, and great weight reduction should not be brought on too precipitately. These patients require the most careful symptomatic management and should be hospitalized during the entire active treatment.

The comparatively rare case with forebrain lesion is the most difficult to treat. No remedy or procedure or dietary regime will effectively control the excessive bulimia or stay the excessive gain in weight. The hyperactivity can be controlled by heavy sedation.

The simple exogenous obesity, fortunately the most frequent and common type at puberty, is the one most amenable to treatment. Basically, the treatment must effect significant reduction of caloric intake and an increased expenditure of energy through exercise. If this program is wisely planned and carried out with complete cooperation of the child and his environment, the result will be certain and the cure permanently effective. Reduction of caloric intake and prescription of exercise will depend on the degree of obesity. It must not reach the point where tissue protein must supply energy needs. There is danger in too aggressive reduction with great loss of tissue protein. Anorexia nervosa in such cases is sometimes a dangerous and formidable complication and will present a far more baffling problem of treatment than the original obesity. There always should be an adequate amount of vitamins and minerals in any reducing diet. If the basal metabolism tends to the lower

normal levels, it is well to give thyroid extract in addition to the dietary regime. It speeds up the metabolism, is in no way harmful, and accelerates a good therapeutic result. The psychologic and psychiatric aspects of the case should be well appraised and well managed. Successful treatment of the case is not at all certain unless this is done. With good, intelligent management, the successful therapeutic outcome of the exogenous obese child is assured, and the problem, physical, social, or otherwise, which it might create, can be cleared up completely. Fortunately, it is the type most commonly encountered in everyday practice.

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ATHLETIC ACTIVITY AT PUBERTY, WITH SPECIAL REFERENCE TO THE CARDIAC AND TUBERCULOUS PATIENT

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THIS extensive symposium on the adolescent child might lead one to suspect that his problems are so many and so varied that he has little chance of coming through this period without being scarred for life, if, indeed, he survives at all. He has growth troubles, endocrine troubles, home troubles, school troubles, and sex troubles. Now that these various phases of the adolescent period have been considered, it remains for me to undertake the prosaic task of attempting to decide whether the child at puberty should be allowed to use the muscles which nature gave him.

The first thing I should like to emphasize is that in any such discussion as we have undertaken, we would do well to remember that the acute and distressing problems occur in only a relatively small percentage of individuals, but, because of their seriousness, they attract an unusual degree of attention. On this account medical opinion is likely to be focused upon the exceptional child, and unless one is cautious, he is prone to interpret the extremes occasionally encountered as being more or less the usual reactions at this age.

What is the problem of the child at puberty as concerns athletic activity? Is this period of rapid growth and development so trying that he must abstain from the competitive games and the various athletic activities in which he has indulged with impunity, and which he has thoroughly enjoyed up to this time? Or should he be encouraged to play to the limit in the belief that a strenuous physical regime will induce a wholesome fatigue, which will in turn, render less acute the problems of emotional and social adjustment? I think one should stress first of all that in considering the child's athletic program, it should be considered in relation to his other activities. What about his social activities? How much does he sleep? What of his nutritional status? Does nicotine or alcohol play a part in the picture? It will often be found that the program as a whole is too heavy, and that the child who is apparently unable to stand the athletic activities usual to his group is not physically fit, even though he may be physically well endowed.

But even when extraneous factors are eliminated, it will be found that a certain small percentage of children are unable to carry out the prescribed athletic program which is tolerated with impunity by the majority. On investigation it is often found that these children are slender, narrow chested, and poorly muscled. They are by nature unable

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to stand the wear and tear of athletic competition, or even of any prolonged physical activity. Such children should obviously receive special consideration and not be driven to undertake athletic activities which are beyond their strength.

I should now like to call attention not to the child who is unable to exercise, but to the child who thinks he is unable to exercise. In my opinion there are a good many children who fall into the group. The symptoms are highly important and frequently misleading. I refer to that group of symptoms which has been variously labeled as effort syndrome, soldier's heart, or neurocirculatory asthenia. The symptoms simulate those of organic heart disease although the physical findings fail to revel any cardiac pathology. There are complaints of palpitation, dizziness, faintness, shortness of breath, and precordial pain following mild or moderate exercise. The signs include forcible heart action, sweating, particularly of the hands, ready flushing of the face, and an anxious expression. This symptom complex was first described among soldiers during our Civil War and was studied extensively by Sir Thomas Lewis and others during World War I. Although this ailment occurs more frequently and in more severe form under the trying conditions of military life, it is by no means uncommon in civilian life. Young adults are most frequently affected, but a considerable percentage of cases occurs among adolescent children. Although the symptoms and signs are chiefly referable to the circulatory system, psychiatric studies have shown that the victims of neurocirculatory asthenia are more or less emotionally unstable. They are often found to be unusually submissive to authority, or, on the other hand, to resent all authority. They do not stand up well under psychic strain. They are subject to fears and anxieties. It is hence doubly important that one should avoid any faint suggestion that the child is suffering from actual cardiac disease. Unfortunately such a diagnosis is too often made. A child at about the age of puberty presents himself to his physician with the story that he is unable to carry out physical activity to which he has been accustomed. He tires out at basketball or gymnasium exercises and is breathless even on climbing a flight of stairs. He easily becomes dizzy and faint. His heart beats rapidly, and he is conscious of its action, perhaps even experiencing a dull ache over the heart. On physical examination the heart is found to be rapid, the apex thrust is forceful, and there is perhaps a short systolic murmur over the precordium. All too often under such circumstances a diagnosis of organic heart disease is made. To make such a diagnosis where only a functional disturbance is present is a serious error. The patient is already convinced that he is ill. To be told that he has heart trouble will go far toward making him a chronic invalid. It goes without saying that a painstaking examination is essential to rule out organic heart disease, tuberculosis or other chronic infection, or hyperthyroidism. Once such conditions have been ruled

out, he should receive a thorough explanation of the nature of his symptoms. He should be told that he has no cause for worry, inasmuch as there is no organic disease. He should be encouraged to exercise as much as he is able without undue fatigue. Often with gradually increasing physical activity, the patient feels better, and after a short period, he is able to resume again a reasonably full schedule of athletic activity. Equal or even greater attention must be given to his emotional problems, which in most instances set the stage for the physical manifestations. Neurocirculatory asthenia is frequently overlooked in the adolescent patient for two reasons; first, the possibility of its occurrence in this group is often not appreciated; and second, the symptoms in the milder cases may be so few as to render its diagnosis difficult. It deserves greater attention, for it is indeed gratifying to be able to rehabilitate these young patients who are convinced that they are suffering from organic disease of the heart.

Let us now consider the problem of the child who does have cardiac damage. At the age of puberty there are two chief types of heart disease which require consideration. These are congenital heart disease and rheumatic heart disease. The patient with congenital heart disease seldom presents a problem. His ailment has been present since birth. Whatever handicap is present is a constant one, due not to infection, but to alteration of function resulting from arrested or distorted development of the heart or vessels. Because this handicap has been present since birth, the patient has learned his physical limitations and makes no effort to exceed them. The avoidance of complicating infections which might of themselves prove fatal or which might induce a fatal bacterial endocarditis at the site of the congenital lesion is the chief consideration in the management of the congenital cardiac child at the age of puberty, or, indeed, at any age.

Rheumatic heart disease is a more frequent and serious problem. For a proper appreciation of the significance of rheumatic heart disease during adolescence, it is necessary to understand the behavior of the rheumatic infection in earlier life. According to statistical data, rheumatic fever usually begins in childhood, occurring most often between the ages of 5 and 12 years. The manifestations may be varied, including one or more of the recognized rheumatic phenomena: polyarthritis, chorea, rheumatic erythema, subcutaneous nodules, or carditis. Whatever the mode of onset, or however many manifestations may occur, the one important feature is the state of the heart. Unfortunately the heart shows definite signs of injury in a high proportion of cases of rheumatic infection, varying in different series from 50 to 80 per cent. Once the heart is involved, recovery is slow, being a matter of months and sometimes years. Even when convalescence has been established, recurrences are prone to occur after a quiescent interval, each attack inflicting increasing damage on the heart. Hence it often happens that a child

approaches the age of puberty with a heart so damaged that he is unable to undertake any strenuous form of exercise. Shall we allow him to do as much as he feels able to do without undue fatigue? It is my belief that we should, provided we can be sure that he is free of active rheumatic infection. That is the crux of the entire matter. Bouts of active infection tend to decrease in frequency and severity at the age of puberty, but they do occur. Because the recrudescences of infection are less stormy, they are more likely to escape observation. The child who is complaining of fatigue on slight exertion, who is a trifle pale, whose appetite has fallen off, who has lost a little weight, or who has an occasional slight fever should be studied by every necessary physical and laboratory means to determine the presence or absence of rheumatic activity. If rheumatic activity is found to be present, the prescription should not call for limited physical exercise. It should call for rest in bed until all evidence of active infection has disappeared. It has long been my contention that we make two mistakes in the management of the juvenile rheumatic patient. In the first place we allow him some degree of activity before the rheumatic process has subsided, and clinical experience has shown that increasing the work of the heart suffering from active rheumatic infection causes a greater degree of permanent damage. In the second place we restrict more than is necessary the patient who has completely recovered from rheumatic fever. The mere presence of valvular injury or some degree of cardiac enlargement does not justify a rigid regime which renders the patient needlessly unhappy. We should recommend a way of life which is temperate and which includes as much activity as can be undertaken without undue fatigue.

Now let us return once more to the question of effort syndrome, or neurocirculatory asthenia, which we have pointed out to be a not infrequent occurrence at about the age of puberty. If the sensitive child who has no cardiac disability is likely to manifest symptoms and signs referable to the circulatory system, how much more likely is the cardiac patient to fall a victim to symptoms and signs of cardiac embarrassment even though his heart is altogether competent for ordinary activity. The child has been told that he has a damaged heart. He has been frequently examined. Perhaps the nature of his cardiac lesions has been discussed in his presence, before other physicians or groups of medical students. He has been asked whether he is short of breath, whether his heart pounds, and whether he has any precordial pain. Is it any wonder that a sensitive, apprehensive child may manifest symptoms and signs pointing to poor cardiac function even though the heart is standing up perfectly well? Under such circumstances it requires the most painstaking study and the nicest discrimination to determine whether the child's symptoms are due to effort syndrome or to the cardiac lesion itself. In the first case he needs reassurance, encouragement, and grad-

ually increased physical activity. In the second case he needs to be restricted in his program, or perhaps put at complete rest.

Another problem which is in many respects similar to the management of the adolescent cardiac patient is that of the tuberculous individual. Many striking similarities are found in the behavior of tuberculosis and the rheumatic infection. Both are chronic ailments. Both may flare into activity after long periods of apparent quiescence. Both are encountered more commonly among those who are unfavorably situated economically. Both tend to be protean in their manifestations. Both may be so stealthy in their attack that the presence of activity may remain in doubt after the most careful physical examination and laboratory tests. It is my firm conviction that the management of the tuberculous patient at puberty is essentially the same as that of the rheumatic patient. If we can be sure that active infection has ceased, the child may be allowed free physical activity. If we know that even the mildest sort of active infection continues, complete rest should be enforced. It is often stated that the adult can conquer a mild tuberculosis standing up. Certainly this is dangerous doctrine for the rapidly growing adolescent child. In fact, pathologic examinations have shown the lesions at this age to be so rapidly advancing that some have spoken of adolescent tuberculosis as a separate entity, noted for its malignancy. Perhaps more accurate would be the recognition of the fact that tuberculosis advances more rapidly when the resistance of the host is low, and that the stresses of the adolescent period, including rapid growth, social activities, loss of sleep, and other irregularities, furnish such a decreased resistance.

In conclusion I should like to stress that no hard and fast rules can be laid down to govern athletic activity at puberty. The exercises which are adapted to the average child may prove too strenuous for the child whose physical endowment has been inferior to that of his fellows. He cannot maintain the pace and should be given special consideration. The nervous child whose symptoms manifest themselves in lowered tolerance for physical exercise must also be shielded from undue exertion.

When we consider athletic activity for the patient with rheumatic heart disease or tuberculosis, our prime objective is to determine the presence or absence of activity of the infection. If activity is present, the immediate objective is complete rest. When activity has ceased, reasonable physical exertion not only should be permitted but should be encouraged. It makes for a healthier state of both mind and body. It goes without saying that these victims of organic disease should receive adequate amounts of rest and sleep and the most wholesome general environment that can possibly be furnished. By such a regime, active infection is less likely to recur, and it is the recurrence of active infection which maims and kills.

SEXUAL EDUCATION OF THE ADOLESCENT

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IN A twenty-minute paper the subject of the sexual education of the adolescent can be dealt with only sketchily. Preventive medicine has made great strides, and often the fruits of much painstaking research can be put into the hands of the practitioner within the confines of an ampule. He may forthwith be able to prevent or greatly minimize dangers that hitherto have taken great toll. Our increased understanding of the mental life of children has enabled us to accomplish considerable in the preventive field of mental hygiene and training. No formula, no ampule, no brief paragraph suffices, however, to permit effective application of this understanding.

Whether or not a special "problem" of "sexual education" exists at adolescence is dependent upon the nature of the child's previous education and social experience. Many adolescents do not need "educating" in this field if by this we mean the imparting of knowledge having to do specifically with sexual behavior and procreation. Every adolescent, however, still has much to learn that is fundamental to an understanding of personal and social relationships.

Certain general attitudes and characteristic emotional states are presented by adolescents. The adolescent is driven to the development of a variety of defensive reactions, designed to help him cope with a quantum of physiologically augmented impulses and urges. He may or may not become consciously aware of the nature of these impulses. These stronger impulses now distinguish his situation from the more placid life period extending roughly from the age of 6 to early puberty. The unpredictable and extremely unstable reactions of many adolescents can best be understood as representing more or less successful attempts to deal with and control the augmented drives.

One fairly common reaction of the adolescent, but not a particularly healthy one, is to deny or to attempt to turn completely away from the existence of sexual interests or of feelings that might lead him into difficult waters. We then see the excessively idealistic, or the more or less ascetic youth, whose activities and behavior carry him into fields apparently far removed from any connotation of sexual interest or activity.* Or the even more common defense of an overemphasized intellectualizing attitude may be encountered. The adolescent here turns his interest and attention to philosophical speculation, to the more abstract social considerations. He may become quite irritating to his

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*For a discussion of the psychology of the adolescent, see Freud, Anna: The Ego and the Mechanisms of Defense, 1937, London, The Hogart Press, Chapter XI.

elders because of his disinterest in practical affairs and his apparent overevaluation of more esoteric interests.

Another possibility is that the adolescent will put up no defense against urgent strivings, or that such defense as he does manifest is swept away. We then must deal with the adolescent delinquent who freely acts out his impulses.

The goal of not only sexual education but of all education of the adolescent is that of enabling him to cope effectively with the turbulent quantum of energetic feeling that tends to overwhelm him. A successful outcome of his struggle is one in which he is driven neither to complete renunciation of sexual strivings, as with the ascetic above mentioned, nor to the undisciplined acting out that characterizes the delinquent. Healthy adolescent experience represents a realistic compromise.

The sexual education of children has proceeded some considerable degree before adolescence is reached. Ideally, before adolescence is reached, children should be quite fully oriented and informed about anatomic differences between the sexes and the normal functioning of all the bodily systems, including the generative organs. This knowledge may have been imparted incidental to general instruction in anatomy and physiology, but in any event it should have proceeded to the point where there is a clear conception of ovulation, fertilization, pregnancy, intrauterine development, and birth. Menstruation, too, should have a rational explanation acceptable to the child.

If this much information is actually understood by the child, an adolescent is likely to have little or no difficulty in frankly raising issues with parents or educators when puzzling or disturbing considerations arise for him. Also, an adolescent so informed accepts a rational discussion of practical issues relating to sexual interest or conduct with a minimum of resistance or anxiety. An adolescent who is *not* thus informed certainly should be instructed in the same spirit and manner in which he is given information in any important field of interest. If this is done, he will then raise the particular issues more personal to himself, which reflect his own unanswered questions, his particular theories, and like as not, his particular anxieties related to sexual interest.

It need hardly be said that in discussing anatomic and physiologic matters with a child, language that he can understand and utilize should be employed. Correct scientific terms should be used, but their colloquial or vulgar equivalents, known to be current among the group with which a child is associated, should also be defined. There is no information having to do with normal sexual relationships, conception, and childbirth that need be withheld from an adolescent. Such information can always be imparted in a manner that does not tend to disturb or stimulate the adolescent unduly. The informant must be a sufficiently mature and experienced person to be able to maintain an appropriate teaching attitude and a comfortable relationship with the adolescent.

The prudent informant, perhaps the physician, will trouble himself to discover first, as fully as he can, just what the adolescent knows and what his experience has been. He can then orient himself accordingly. If the physician has the confidence of the adolescent, the latter, with a little help, is usually capable of stating what he understands. The simple expedient of having a youth state how he would explain about conception and childbirth, if he had to be the informant, generally is revealing. The well-informed adolescent who is in no great conflict will be able to give straightforward statements. The inhibited or anxious adolescent may be able to give little factual information, but will express his attitudes and uncertainty. This opens the way for the physician to be of help to him.

Some of the questions that ordinarily arise in clarifying sexual issues for the adolescent call for particularly good judgment. There may be danger of unduly stimulating latent interest or perhaps of being unwittingly too permissive. In dealing with the problem of masturbatory activity, no good purpose is served unless anxiety is alleviated. At the same time, a too permissive attitude tends to break down the normal defense against this childish activity. Here good judgment about the character of the individual, his capacity for healthy activity and accomplishment, or his tendency toward withdrawal and isolation will determine the emphasis made by the physician. It will be in the direction of reassurance in dealing with an anxious child burdened with guilty feeling about masturbation. Control and the desirability of giving up childish behavior will be emphasized with a child who freely acts out his impulses with little or no conflict.

It cannot be assumed, however, that rational descriptions and explanations given a child necessarily will have the meaning to him that the informant intends. At least two important considerations may interfere in what is expected to be a simple process of learning. One of these is inherent in the attitude of the parent or informant. An inability to be clear, concise, and forthright, a necessity to smother simple facts behind ambiguous terminology, or possibly sentimental, unrealistic, or overidealistic attitudes on the part of the informant may make it quite impossible for the child to gain understanding. Such defensive tactics on the part of the parent are based primarily upon his fear of having the child adequately informed. A situation in point is that of an adolescent girl, who, according to the mother, was given full sexual information by the mother herself, some time before the girl was to go away to an excellent school for girls. She returned to her home at the Christmas vacation period, reported to her mother about the "bad" talk she had overheard among the girls, and rebuked the mother for having sent her away from home so uninformed in matters about which the other girls seemed to know a great deal. She was unable to connect the instruction given her by the mother with the frank talk about boys

and sexual matters current among the girls in the school. Young people in late adolescence, particularly girls, will at times comment upon the fact that their parents gave them absolutely no sexual information, even when this is not in accord with the facts. In one instance, a young woman made similar comment although the parents actually had sought the aid of a competent physician, who had endeavored to inform the child, shortly before the age of pubescence. An amnesia apparently exists in relation to information for which the child is unprepared, or situations that are too threatening.

An important consideration then is the great resistance some children manifest toward the assimilation of sexual knowledge. Where a parent is on unsure ground in discussing sexual matters, as in the case above cited, the child is not likely to maintain an undisturbed and receptive attitude necessary for comprehension in any field. Sexual interest tends to be vested with anxiety for most children, and unless this anxiety can be avoided or dispelled, real progress toward an understanding of mature sexual relationships is likely to be remote. The conventional prohibitions and taboos to which the child is subjected are a source of this anxiety. A more important source lies in his earliest attitudes of childhood affection for parents, and resentment, jealousy, and hostility toward those persons, whether parents or siblings, who interfere with an exclusive, possessive relationship with the best-loved parent.

Limitations of time for this paper do not permit elaboration of this important theme. I would nevertheless emphasize that the manner in which the very young child solves the problem of his relationship to the members of his family profoundly influences his social and sexual attitudes during adolescence. The ideal situation is one in which a small child has experienced real warmth and affection and a reasonable amount of actual physical fondling in his home. He develops a certain feeling of self-assurance and self-esteem. Even if the situation is not overdone, for a while he is likely to be somewhat jealous and envious of other members of the family. If he is neither deprived of affection, nor encouraged in a too close or too dependent relationship with either parent, he will normally eventually take the step of allying himself in feeling or in identifying with the parent of his own sex. In so doing, he lays the basis for the later development of normal sexual attitudes. He is then able to react in terms of the proposition "I am a person like my father; I can do and enjoy those things and activities he does and enjoys. I can accomplish the same sorts of works. On the whole, my rights and privileges become like his." Any adolescent who has substantially arrived at this sort of inner feeling is well on the road to the development of a mature personality. We are assuming, however, that the parental figure in relation to whom he has developed such a concept is himself an emotionally healthy individual. The same process,

in relation to a neurotic or criminal father, can only have an unfortunate outcome on the child.

It is when this normal process of development in some way has been interfered with that the adolescent encounters difficulties in intellectually and emotionally comprehending mature social and sexual relationships.

Whether or not the sexual education of the adolescent involves any reference to pathology depends upon the environmental influences to which the individual is exposed and to the practical imminence of the likelihood of actual indulgence in adult sexual behavior. It may be that the particular social environment to which he is exposed spares the adolescent not at all, in that his attention is called to venereal disease, or possibly to abnormal or perverse behavior. Here again it is up to the physician to know the individual and his background sufficiently well that he does not unduly provoke issues that need not yet be raised, nor on the other hand, avoid frank facing of issues that require understanding and clarification for the protection of the youth.

In my opinion, parents should be the persons best qualified to instruct their own children in sexual matters. Many parents, however, are unable to approach this task successfully, as I have already indicated. Under some conditions, schools are in a position to instruct children in this field. This, however, is dependent upon special knowledge, skill, and aptitude on the part of a particular individual or individuals in the school setting. It is not a teaching function to be performed by teachers in general. Parents would have to be cognizant and accepting of such instruction by the school.

Physicians, and most particularly pediatricians who often are acquainted with a child throughout his lifetime, are in a position of particular advantage to be helpful to parents and to children in providing instruction and counsel in this exacting field. Direct discussion of sexual matters with an adolescent, however, cannot ordinarily be undertaken by the busy pediatrician in the course of routine office consultations. Some planning of time, permitting unhurried discussion, is necessary. The investment of time need not be great. *Effectiveness* in the use of this time will be dependent upon an understanding of the basic attitudes of the adolescent and the overcoming of his particular resistances to an acceptance of the information offered him.

PSYCHOLOGIC ASPECTS OF ADOLESCENCE

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OF ALL specialists engaged in the practice of medicine, the pediatrician must be of necessity the most versatile. He must always be ready to shift his point of view or change his perspective. It is essential that he be sensitive to the fact that as his patients advance from one age to another, alertness on his part to the changes taking place is important if he is to take advantage of the prestige which each patient attaches to his particular age. An age difference of two or three years between the ages of 4 and 14 years means something quite different in the physician's approach to his patient from his approach twenty years later.

The pediatrician is in a unique position, by virtue of the continuity of his service, not only to contribute to the solution of problems which arise during the adolescent period, but also to guide and direct the growing child so that many of the adolescent problems may be prevented or at least minimized. In the pediatrician's almost unrestricted field, which begins shortly after the birth of his patient and does not terminate until the "teen" age is well advanced, he may exert a definite stabilizing influence on the future health and happiness of a child and the peace of mind of the parents.

Preadolescent years are, on the whole, fairly well coordinated one with another. Uniformity of growth and of physiologic patterns allows the child to develop without too much awareness of self. His appetites, desires, feelings, and emotions tend to keep pace with his opportunity for exercising these needs. His life is thus fairly well stabilized at the end of the first decade. As the child is sufficiently well acquainted with the organism which he has to manipulate, he is not being constantly subjected to unexpected and baffling experiences.

The adolescent period itself, however, is punctuated by striking evidences of disharmony and incoordination between anatomic growth, physiologic development, and psychologic maturation.

No one, in my opinion, is more cognizant of the importance of growth and its effect upon behavior than the pediatrician. He is in a position not only to observe but also to understand the problem which confronts the 14-year-old who has gained 30 pounds in weight and 6 inches in

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height in a twelve-month period. He realizes that the resulting awkwardness, laziness, and self-consciousness create confusion in the child's mind, and that parents fail to understand that this business of growing up is not the simple process that it may seem. The pediatrician is also aware of the physiologic changes that take place during adolescence and their psychologic implications. He appreciates how these changes affect the emotional life of the child. But like the psychiatrist, he may not always know how or why certain urges, desires, and appetites are so suddenly and unexpectedly awakened in some cases and intensified in others at a time when the experiences of the child are still so limited and when the inhibitory forces which are essential to direct these emotions are so unpredictable. The fact remains that anatomically, physiologically, and psychologically disharmonies do take place. The physician, particularly the pediatrician, is often the one who is called upon to interpret their meaning to both adolescent and parent in order that unhappy consequences may be avoided.

The physician knows that growth proceeds at varying rates of speed in different individuals, and in the same individual at different stages, and he is not unduly concerned with the poor motor coordination which so often occurs. Time, he says, will strike a satisfactory balance between muscular and bony development and all will turn out well, but in the meantime the adolescent's clumsy, awkward behavior often becomes a source of irritation to the family and extreme embarrassment to the adolescent himself. The youngster is nagged, teased, and not infrequently punished for his carelessness, most of which is quite beyond his control. He gets moody, unhappy, resentful toward what he considers unfair treatment and lack of understanding. The aggressive boy is likely to get into social difficulties (minor delinquencies), while the sensitive boy will become more diffident because of his self-consciousness and tend to withdraw from social activities in so far as it is possible. The pediatrician who understands and anticipates the difficulties that are likely to arise in particular children because of these irregularities in the process of growth and who can help parent and child over this trying period is rendering a real service.

Sex, to be sure, is not a problem specific to the adolescent period, yet it is often featured at this particular time. So much has been written on the subject of late and our professional attitudes have so radically changed during the past twenty-five years that we are apt to take it for granted that the layman, too, has followed along and kept pace with the physicians, consequently becoming more objective over the problem, and that children as a group hold a healthier point of view on sex. But such is positively not the case. There never was a time as much as the present when children were in greater need of sound education during

the first decade of life regarding the subject of reproduction, to be followed by a more intimate and detailed discussion of the physiologic aspects of sex and its relation to social development. Such knowledge, as we know, is not acquired by a few hurried formal periods of instruction, but this important aspect of the child's education should be given slowly and in small doses, as the growing curiosity demands, so that it can be well digested and assimilated.

Girls are usually given sufficient information to prepare them to meet the hygienic needs of their first menstrual period, but they are all too frequently left entirely in the dark as to its physiologic and biologic significance. Boys are so infrequently told anything about the anatomy or physiology of the genital system that they are often tremendously worried over their first nocturnal emissions and embarrassed by having an erection; they not infrequently think they have some abnormality because the left testicle is larger than the right.

It can be taken for granted that the problem of masturbation is not particularly different in this streamlined age than it was when we were boys. Parents are still much concerned over its causes and consequences but are quite as inadequate to meet it frankly. This anxiety is passed on to the children and it is the fear and guilt feelings over the habit rather than the habit itself which leave the scars. These children continue to worry about the effect of the habit upon their mentality; they are sure it is the cause of the acne which has been present for months and are convinced that everyone knows of their secret sin. The feelings of guilt and the inferiority which follow affect their social relationships. It is during this particular stage of life that many children become decidedly asocial, shy, diffident, submissive, with occasional explosive outbursts which may bring them into conflict with society. Truancy, stealing, destructiveness are often manifestations of an aggressiveness which is not infrequently associated with conflicts about sex. Aggressiveness appears to serve the purpose of diverting the child's attention from urgent sex drives which are so incompatible with his ideals.

Within the month I have seen two boys whose conduct has been affected by anxieties over masturbation. The fathers of both boys are professional men and the mothers belong to what may be termed the proper social set. Neither of these lads had had any intelligent sex instruction. The 16-year-old boy had a fairly severe depression with a deep sense of guilt over his indulgences. The other, a 14-year-old lad, got into serious difficulty at a private school by disseminating obscene pictures. Notwithstanding, both of these lads were under the medical supervision of a pediatrician. It is my contention that although many of the boys between 14 and 16 years of age feel that they know "all

the answers to most of the questions," it is the responsibility of their medical adviser to see that such information is reasonably authentic.

I am not presuming to tell this group that these problems are important in relation to mental health, nor am I going into detail as to how they should be treated. I am simply pointing out that these problems are a primary concern of the pediatrician. Not only should he recognize the symptoms of morbid anxiety and be able to carry out treatment, but what is more important, he should anticipate these pitfalls and through educational methods prevent these common conflicts from becoming real problems.

We should not forget that parents will not be coming to the pediatrician complaining about Johnny's awkwardness; to them this is a problem of training and discipline to be handled by them as such. Nor will the adolescent boy commonly seek advice about nocturnal emissions, for this is something of which he is both afraid and ashamed. The pediatrician must take it for granted that as a rule parents are unmindful, ignorant, or neglectful of this aspect of the child's education and that it is up to him to anticipate the danger signals. Rarely will the adolescent speak first; the doctor must tactfully, seriously, and courageously take the initiative and frankly open up the subject of sex when the occasion demands.

Let us consider for a moment another adolescent problem, which is not less common than the measles during the preschool period, but which causes parents almost universal concern. I am referring to the struggles and conflicts which the adolescent has in establishing his own independence, a very normal and healthy desire on the part of the child but one which is often met by considerable resistance on the part of parents. During this important phase of psychologic development, the parent-child relationship can often be steered away from those dangerous emotional shoals by wise guidance on the part of the pediatrician. The vital point here is for the pediatrician, in the first place, to help the child grasp the idea that freedom, with its new privileges and opportunities, carries with it obligations and responsibilities; and in the second place, to aid parents to grasp the notion that the ultimate objective of child training is to prepare the child to live happily and efficiently in a world that will grant no special concessions to their particular child. Parents must be helped to appreciate that if children are to learn how to utilize freedom wisely, boys and girls must of necessity have enough freedom to make mistakes from which the average child will profit. Who, of all of the family consultants and advisers, should be more adequately equipped by training and be in a better strategic position to guide both parent and child wisely during this important phase of development than the pediatrician?

Let us not forget that many of the relatively simple problems common to the various age groups develop into definite psychiatric situations when the child grows older if they have not been wisely managed when they first make their appearance. This is particularly true in the case of the inherently sensitive child, the constitutionally unstable, and the overaggressive. Parents as a group are dependent upon the physician for guidance not only when children are in difficulties but also in preventing them from getting hopelessly entangled in relatively unimportant emotional situations. Simple as these problems may be in their origin, the relationship between cause and effect becomes obscured by time, and the whole situation may appear very complex and complicated when it eventually reaches the psychiatrist. (This assertion is based upon an experience as a consultant psychiatrist to a large private school for boys and one of the outstanding New England colleges for women and in private practice with varied groups of neurotic and delinquent children.)

Many children become the victims of their parents' ambitions or their failures and disillusionments; others get entangled in the emotional frustrations and disappointments to which parents have been subjected. However, the most common cause of an unhappy parent-child relationship is just a plain lack of understanding which the average parent has of the emotional needs of a child. The failure to appreciate that a child has a mental life which must be nourished and nurtured and not simply directed and dominated creates quite as many problems as sheer parental neglect.

The applications of mental hygiene to the fundamental problems of growth, sex, and independence have been discussed in a very general way. A brief consideration will be given in the following discussion to a few of the more specific problems which the pediatrician encounters during the adolescent period.

Problems of an educational nature are not infrequently precipitated by change from the elementary to the high school. Up to this time many children are often passed along in both private and public schools without being adequately prepared to meet the scholastic demands of the grade to which they are promoted. In public schools promotion is often a matter of necessity to make room for the incoming group. In the private school promotion may be purely a matter of expediency to keep the family satisfied, so with extra work in school and with summer tutoring the boy is kept moving, usually dragging along at the bottom of his class.

There are, of course, the common disabilities which account for school failure. The two which are encountered most frequently are poor mental equipment and reading handicaps, both of which must be de-

termined by psychologic tests which should be given by a clinically trained psychologist. When such handicaps exist, an effort should be made to place the child so that such capacities and abilities as he may possess will be recognized and trained.

Children with special disabilities need special training; re-education should be carried along whenever possible without interrupting the normal course of their school life, and children should not be withdrawn from their routine activities unless absolutely necessary and have their attention focused on their disability to the exclusion of everything else.

However, many children with good intellectual equipment and no particular educational handicaps have difficulty with their school work and not infrequently their problems are rather acute. One parent reports that until John entered high school he did good work and got high marks; in fact, he got by his first year in high school very satisfactorily, but for the past two years his work has steadily fallen off and he is no longer the responsible, obedient boy he used to be. What is the answer?

This particular boy was a professional man's son. About the middle of his freshman year in high school his brother, who was two years older, died quite suddenly. He was a brilliant, all-round, capable boy in whom both parents were very much tied up emotionally. Their reaction to the boy's death was extreme, and the life of the parents was soon built up around their sorrow and the dead rather than the patient. Every anniversary became a day of mourning, whether it commemorated the first day the boy went to school or the day of his death. The parents never entertained or went out, and the patient was constantly being reminded that he should not be so frivolous, considering what had just happened to his brother. Briefly, the boy's behavior problems, which were reflected in his school failure, were simply a revolt against the parents' neglect and their shortsightedness in building the home around the memory of the dead rather than the needs of the living.

Mary was a 15-year-old girl sent to me from the nerve department of a general hospital because she had been suffering for thirteen months with an hysterical aphonia. Her sister gave the following history: The patient had been an excellent student until the time she entered high school. During her freshman year her work was good, but several of her friends forged ahead and the patient began to be unhappy and discouraged. She developed an acute laryngitis and at her own request was excused from reciting. This condition dragged along unduly until the teacher finally told the patient that the following Monday she must begin to recite and take her turn with the other pupils. When Mary arrived at school the beginning of the following week, she developed a complete

hysterical aphonia which, as previously stated, lasted thirteen months and took her out of school. This is a rather interesting and spectacular way of meeting a difficult situation. The aphonic condition freed her from an intolerable situation of having to meet competition and face what to her meant certain failure.

School phobias are rather common and are more frequently seen in girls than boys. Within the past year I have seen one child protecting herself from an unpleasant school situation with neurotic vomiting, another with fainting spells, and a third with attacks that simulated asthma. Two of these children had absolutely nothing organic to account for the vomiting or fainting but a very definite psychologic reason for evading school. The child with asthmatic attacks had a father who had violent paroxysms due to asthma about which he got very hysterical, and the child herself had had earlier in her life a history of asthma. In her case, however, her recent attacks were precipitated by the threat of being sent to school.

I have merely mentioned three outstanding, almost universal, phases of adolescent development that naturally come to the attention of pediatricians, without any intent of questioning their ability to cope with these problems. Yet I venture to say that the knowledge they have acquired in dealing with these problems wisely has been the result of practice rather than of professional training.

Up to the present time neither undergraduate nor postgraduate study in pediatrics in many of our medical schools makes adequate provision for training students in mental hygiene. I attempted to evaluate with a pediatrician the training given in twenty-eight medical schools where the head of the department in pediatrics was kind enough to send me a statement covering the teaching of mental hygiene in relation to pediatrics. In seven schools the plan was adequate; in nine it was fair; and in twelve it was poor. All too frequently students leave medical school scarcely aware of the existence of psychologic problems which are, as we have seen, directly related to pediatrics. Unfortunately, they learn to consider these problems not as normal, natural behavior responses that occur in the process of growing up but as peculiarities and eccentricities of human behavior and, therefore, as belonging to the field of psychiatry.

Pediatrics is concerned with growth and development, but as taught in many of our medical schools, it is limited to considerations of physical growth and intellectual development alone. The emotional aspect of child life and social development and the whole problem of personality have been all but ignored until recently. It is only after years of practical experience and many unnecessary mistakes of both omission and commission that the pediatrician arrives at the stage where he gives

adequate consideration to those inherent and environmental factors which affect mental health. Considerable information pertaining to mental health is available which is sound, practical, and teachable but is not being utilized. It is not unusual that the pediatrician, when he embarks upon the practice of medicine, has an accumulation of knowledge pertaining to mental hygiene gathered from psychiatry which is so poorly coordinated with this pediatric knowledge that it is practically useless. This is due very largely to the fact that mental hygiene as it relates to pediatrics has not been taught in medical school and during hospital training in such a way as to impress the student with its practical value. Consequently the pediatrician's potential value to his patients and their parents is often not fully realized until several years after graduation. The result is that many children flounder through very critical periods in life without receiving from the pediatrician the assistance to which they are entitled.

There are several reasons for this situation. First, there are pediatricians in practice who say, "Perfectly true. This is just common sense and it is what we all do." To this group I would say, "It is my feeling that your intentions are good, that you may well comprehend the needs of both parent and child, but that you fail to appreciate that common sense is not a common commodity." Second, there is another group who say, "This all may be true, but it is not my job; it's too time-consuming," and leave the responsibility to somebody else. Third, there are pediatricians who evade the issue by minimizing the importance of the psychologic aspects of physical problems. Baffled and confused by the inadequacy of their anatomic, physiologic, and biochemical knowledge to combat these psychologic problems, they become annoyed and irritated and deny the existence of the problem. Fourth, there are pediatricians who understand the psychologic mechanisms involved but find it extremely difficult, because of personal emotional blocks, to delve into sex and personality problems involving either parents, or children, or both. They wisely refer these cases to those in whom they have confidence.

Fifth, and finally, there are those pediatricians who have avoided psychologic treatment because some psychiatrists have tended to shroud the mental life of the child with incomprehensible mysticism. Some therapists (belonging to the analytical school) would have every child with a behavior problem subjected to a long-drawn-out expensive analysis. For example, Dr. Mary O'Neil Hawkins, in a paper read before the meeting of the American Pediatric Association in Kansas City, Mo., speaking of enuresis, stated:

"Psychoanalysis is indicated in all cases of enuresis occurring during late childhood, in spite of the fact that the symptom itself will almost always disappear eventually. This recommendation is based upon

psychoanalytic investigation which has shown that this symptom occurs only when there is a serious disturbance in sexual development. Most of these patients will show some form of neurosis in later life."[1]

The above reasons for not dealing frankly and adequately with the adolescent and preadolescent problems, I am sure, could be overcome to a very large extent through adequate training during undergraduate and postgraduate years which would bring mental hygiene and pediatrics into closer association. The pediatrician who is interested in the emotional conflicts which account for feeding difficulties, temper tantrums, terrifying dreams, shyness, and overdependence during the preschool years is likely to be the one who will contribute to the parents' understanding of adolescent rebellion against authority, secretiveness, aggressiveness leading to asocial behavior, and erratic and unpredictable mood changes.

It is my feeling that mental hygiene and psychiatry as they apply to medicine in general should be taught simultaneously with the teaching of the particular branch of medicine that is being presented. In other words, such contributions as psychiatry has to make should not be looked upon as a specialized discipline that is superimposed after the student has acquired the fundamentals of pediatrics, medicine, or surgery, but rather as integral to those fundamentals. For instance, when the student is receiving instruction in pediatrics and feeding is being discussed from the biochemical and physiologic points of view, the emotional attitude of the child and parent toward food should be given careful consideration. The oversolicitous, aggressive mother who at mealtimes sets the stage whereby the child has an opportunity of exercising his power and successfully gaining attention for himself is conditioning this child to seek personal satisfactions in the dominations of others. This attitude becomes rather serious during the adolescent age and frequently we find these boys or girls either seeking companionship in lower social strata or being entirely without friendships. This is but one example of a rather common pediatric problem which may have serious implications as the child approaches puberty. The unhealthy habits and mental attitudes of the nursery school may become the difficult psychiatric problems of adolescence. Prevention is easier and more practical than treatment.

The point I would make is that the emotional situations which are developed around feeding, for instance, are quite as significant as the physical problems; they are both part and parcel of the same child, the same personality, and the same environment.

Likewise enuresis, as another example, may not be looked upon as a serious calamity. Nevertheless, if it is, it may create a feeling of inferiority toward which the child will continue to react long after the habit has ceased, becoming very much in evidence during the adolescent

years. For instance, enuresis may allow a child to compete successfully for attention with a younger child and encourage him to cling to infantile ways of responding to life. Yet the treatment need not be complicated or prolonged in order to be successful.

There are a multitude of similar pediatric situations which are important to adolescents, the psychologic significance of which should be brought to light in the teaching of pediatrics.

I know of no other way of training the medical student who is to assume the responsibility for the physical growth and emotional development of children during the first decade and a half than to present him with a clear picture of the relationship which exists between disturbances of function and disturbances of behavior. Only by some such method of training can he acquire the necessary understanding of the personality deviations of the adolescent period and learn to treat them wisely. There is considerable literature and many demonstrations of how such training should be carried out as exemplified by pediatricians themselves in several medical schools. There is need, however, for such training to be extended.

The mental health of children as a group is largely dependent upon practices and precepts of the home, the intelligence rather than the intellectuality of the teachers, and the wisdom, breadth of vision, and training of their medical advisers. When the problems of growing up are recognized and treated at their source by the simplest methods that are essential to solve the situation, then, and only then, will children benefit from our present-day concepts of mental hygiene. The pediatrician is in the front line of defense to protect the child against psychic trauma as well as physical disease.

It is my feeling that pediatricians probably would agree that the relationship between physical and mental development is such that both aspects of this process of growth should be looked upon as being more or less mutually dependent one upon the other. The child's physical development may be materially affected by a long-continued, stressful, emotional relationship with parent, and as has already been shown, many emotional problems occur when physical growth fails to keep pace with chronologic years. It matters not whether it lags behind or rushes ahead. It is out of step and a situation is created. This being true, nothing but confusion can arise in the mind of the student if the pediatrician leans too heavily on the psychiatrist for explanation of how physical problems affect behavior. The pediatrician should take a more important part in impressing the student that deviations in behavior and emotional disturbances, which are clinically looked upon as habit, and personality disorders are found in so many of the general run of cases, in varying degrees of severity, that they are not oddities and peculiarities but commonplace evidences of the whole problem of growing up.

The interpretation of pediatric behavior problems in terms of psychiatric concepts rather than in terms of personality development, which is but part of the process of growing up, seems quite unnecessary. The importance of the pediatrician's doing the mental hygiene teaching and not turning the job over to the psychiatrist cannot be overemphasized. The pediatrician has been disposed to hire someone else to assume a responsibility that is definitely part of his job.

The psychiatric disciplines which are necessary for a pediatrician are quite different from those essential for one who would pursue psychiatry as a specialty. Students should be subjected to a training which gives adequate concern to behavior as well as structure, and they should be influenced to interpret the success of their influence in terms of the child's adaptations to life as well as anatomic and physiologic soundness and efficiency.

No less important is the need of impressing upon the psychiatrist and especially the psychiatric social worker the fact that behavior is not an isolated and quite independent part of the child's life but is closely related to the physical organism and its well-being.

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THE USE OF BOVINE ANTITOXIN FOR THE PROPHYLAXIS OF TETANUS

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THE death rate from tetanus, according to Moore and Singleton[1] who studied the distribution of this disease in the United States, has been reduced one-half during the past fifteen years. The disease, however, still causes more than 1,000 deaths annually. It has been pointed out by Cooke[2] that approximately 50 per cent of all deaths from tetanus occur in children under 15 years of age, so that the disease is at least as important to the pediatrician as to any other medical specialist. In spite of its actual rarity, however, tetanus is such a horrible disease and the prognosis is so poor that the thought of the possible occurrence of tetanus is uppermost in the mind of the physician treating a wound where the indications for the administration of a prophylactic dose of tetanus antitoxin may be present. This communication, however, is not concerned with the necessity for giving tetanus antitoxin, its dosage, or the indications for its use. For these, reference is made particularly to the work of Gilles,[3] Tenbroeck and Bauer,[4] and of Spaeth.[5]

In any discussion of the prophylaxis of tetanus, consideration must be given the use of tetanus toxoid. It is not within the scope of this communication to review the now extensive literature on this subject. However valuable tetanus toxoid may be for mass prophylaxis, especially in the armies of the world and in certain special occupations, it is not practical for routine use in the practice of pediatrics, and particularly in private practice, where the pediatrician has an intimate and direct responsibility to his patient and the family of the patient. The reason for this is that there is no simple procedure, and, indeed, no present procedure except a very complicated and expensive laboratory method, to determine whether or not adequate protection has been conferred by tetanus toxoid. Until some simple test for immunity to tetanus as reliable as the Schick test for immunity to diphtheria has been devised, the physician treating with toxoid a patient presumably exposed to tetanus is knowingly taking a chance with his patient's life, a chance which is certainly much greater than when tetanus antitoxin is used as a prophylactic measure. It is for this reason that Ely[6] in reviewing the use of tetanus toxoid makes the following statement: "Therefore, if a patient who has previously received . . . toxoid is wounded under circumstances indicating that tetanus spores are likely

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to be present in the wound, it is better to give not only . . . tetanus toxoid, but also a prophylactic dose of tetanus antitoxin." If this is true, then there is no particular point to the statement that the presence of allergy in an individual is an indication for the routine use of tetanus toxoid so as to avoid the possible necessity of injecting serum in the future.

The great objection to the use of antitoxin in the prophylaxis of tetanus has always been fear of the unfortunate sequelae which may follow the injection of the commonly used antitoxin derived from horses. Practically all authors considering the subject acknowledge the truth of this. Calvin[7] expresses this opinion as follows: "The chief objection to the prophylactic treatment of tetanus with antiserum is the high incidence of serum sickness which develops five to ten days after the injection of tetanus antitoxin, even after the small amount needed for prophylaxis, not to mention the danger always present of immediate anaphylaxis to horse serum." In addition to this might be mentioned the frequent sensitization of the individual to subsequent injections of horse serum.

When I started practice, I was particularly interested in the field of allergy as applied to pediatrics. Because of this interest in allergy it was only natural that the major part of my practice should be devoted gradually to the care of allergic children. My experiences with tetanus antitoxin derived from horse serum were consistently disagreeable. In 1933 my attention was directed by Dr. James S. Houck to the fact that a commercial tetanus antitoxin made from bovine blood* was available in this country, and since then I have used no other preparation in my practice.

Bovine antitoxin for the prophylaxis of tetanus and diphtheria was introduced by Kraus and his associates in Buenos Aires in about 1921 and was introduced into this country in 1929. The sparse literature of the therapeutic use of normal bovine serum is reviewed in their communications. Kraus was led to the use of bovine serum for specific therapeusis because of experiences in giving large amounts of normal bovine serum in a large series of cases of human anthrax. Of 290 patients with anthrax given 10 to 200 c.c. of normal cattle serum subcutaneously or intravenously, in only six patients, or 2.1 per cent, did serum sickness appear, and then only mildly, the manifestations being limited to a rash without fever or arthralgia. In forty cases of typhus fever treated with normal cattle serum, no cases of serum sickness occurred. In forty-six cases where the cattle serum was reinjected in doses of 10 to 30 c.c. one to four weeks after a primary injection of 100 to 200 c.c., immediate

*All of the bovine tetanus antitoxin used by the author was purchased on the open market and was prepared by the Mulford Biological Laboratories, Sharp & Dohme Co., Philadelphia, Pa. As far as the author can determine, no other bovine tetanus antitoxin is prepared in the United States or Canada. Until the lyophil (Lyovac) preparation, which has a five-year dating, became available in 1939, the conventional liquid serum was used. The lyophilized tetanus antitoxin (bovine) is available in 1,500-unit and 10,000-unit doses. Each package contains a 1 c.c. ampule of normal bovine serum (diluted 1:10) as test and hyposensitizing material. If the preparation becomes outdated before use, it will be exchanged by the company without charge.

reactions occurred in two cases, one consisting of an exanthem and fever for one day and the other of joint pains; two other cases cases developed delayed serum sickness, a total incidence of serum reactions of 8.7 per cent. Alarming anaphylactic reactions apparently did not occur in any instances. The striking absence of allergic reactions to bovine serum suggested the use of such serum for the preparation of therapeutic antiserums, especially for tetanus and diphtheria, which were at that time and are still largely the principal diseases treated by sera. It was found that horses produced better antitoxin than cattle, but concentration of the serum compensates for this slight disadvantage which is insignificant in view of the safety of the bovine serum. Kraus[8, 9] treated forty-nine cases of diphtheria and 149 patients prophylactically. Serum sickness occurred in only nine instances, or 4.54 per cent. Similar results were reported with the use of bovine tetanus antitoxin. The authors concluded that just as good results could be obtained with bovine serum, and that by its use the disagreeable reactions caused by horse serum may be avoided. Since the work of Kraus and his associates, there has been but little mention of the therapeutic use of bovine antitoxin. Hanke[10] in 1933 and Graf[11] in 1936 treated anthrax with bovine serum. In 1933 Fleisher and Jones[12] observed that beef serum was less active than horse serum in producing serum sickness in rabbits. In 1936 Glaser and Landau[13] advised the use of bovine tetanus antitoxin with particular reference to the avoidance of serum reactions and sensitization to horse serum. More recently Wangensteen and his associates[14] have reported that bovine plasma may be given intravenously to man in fairly large quantities; it is retained and apparently utilized. In a series of sixty-six patients receiving 2 to 10 c.c. intravenously, reactions were noted in ten cases, or 15 per cent. Nine gave mild to moderate reactions. There was one severe reaction in a patient with a history of asthma.

My experience with bovine tetanus antitoxin consists of thirty-eight cases. The ages of the patients were from 1½ to 18 years, with an average age of 7 years. The essential details of the cases are recorded in Table I.

Special interest is attached to Cases 5, 22, 33, and 36, and these will be discussed briefly before an attempt is made to summarize the data in Table I.

CASE 5.—This boy was admitted to the emergency treatment room of the Genesee Hospital for treatment of numerous brush burns and scratches received as the result of an auto accident in which he had been dragged for a distance along the road. The attending physician advised tetanus antitoxin, and the boy was given a routine intradermal test with horse serum diluted 1:10. Immediately following this he became cyanotic and unconscious and made convulsive movements, and it was thought that he was going to die because of immediate anaphylactic shock. However, he revived upon the subcutaneous injection of epinephrine hydrochloride, 1:1,000 solution, and with artificial respiration. He was kept in the hospital over night and referred to the Allergy Clinic where he appeared a few days later.

At this time he was tested with 1:10 bovine serum intradermally, with negative results (following a negative scratch test to the same substance), and 1,500 units of bovine tetanus antitoxin were given in divided doses over a period of one hour, with no difficulty whatsoever. The boy was detained at the hospital for several hours before being sent home. Two weeks later he reappeared at the Clinic and reported no symptoms which might be interpreted as a serum reaction. This was the only case which was not treated in private practice, and, coming early in the series, it impressed upon all observers the value of bovine tetanus antitoxin.

CASE 22.—This boy had had asthma since the age of 2 years. At the age of 8 years he came under treatment which was discontinued against advice at the age of 11 years, having been completely free from attacks the last year of treatment except for two instances when an adequate and easily avoidable exposure caused mild attacks. On July 4, 1940, while visiting in a neighboring town, a firecracker exploded in his hands, severely lacerating the fingers and palms. The wounds were treated by a local physician who also attempted to administer horse serum antitoxin despite a strongly positive skin test. The patient was given two injections of an unknown but small amount at half-hour intervals. One-half hour after the second injection, he experienced itching about the eyes, broke out with generalized urticarial wheals, and complained of abdominal pain and difficulty in breathing. The symptoms were relieved by the administration of epinephrine hydrochloride, and the boy was brought back to Rochester. The following day he was somewhat edematous and asthmatic and suffered from nausea and vomiting. A day later he was given a scratch test to bovine serum, and this was followed by a negative intradermal test to the same substance. Fifteen hundred units of bovine tetanus antitoxin were then given in divided doses, without difficulty. Two days later he experienced a severe delayed reaction to the previous injections of the horse serum. This was evident because of several local reactions at the site of the previous skin test to horse serum and the two sites where the small amounts of horse serum had been injected. There was no reaction whatsoever at the sites of testing and injection with the bovine serum. He was uncomfortable for a number of days from this, and when it cleared up about six days after the original injury, he was again given 1,500 units of bovine tetanus antitoxin with no disturbing sequelae then or at any time later.

CASE 33.—This was a boy 14 years of age who consulted Dr. C. V. Costello because of an accidental shot with an air gun, necessitating the surgical removal of the bullet from between the bones of his wrist. Tetanus antitoxin was advised. The parents strenuously objected to this because they stated that at the age of 5 years the boy had been almost killed by an injection of tetanus antitoxin. However, I felt that the bovine tetanus antitoxin deserved a trial in this case, and this was given uneventfully and without any untoward sequelae of any kind.

CASE 36.—This case is somewhat similar to Case 33, but in this instance the parents were reluctant to have tetanus antitoxin administered because an older brother had once had a bad reaction to tetanus antitoxin. The bovine serum, however, was administered uneventfully.

Case 6 was the only patient whom it was necessary to see at home or in the office because of a serum reaction. This boy was seen at home, as he was moderately ill for three or four days following the injection of the bovine tetanus antitoxin. This was the worst serum reaction in the series and, curiously enough, it occurred in a nonallergic individual. Case 28 was not seen after the injection, but his mother reported by

TABLE I

DATA OF CASES STUDIED

CASE NO.	AGE (YR.)	SEX	PERSONAL ALLERGY	NATURE OF INJURY	REACTION	DAYS	REMARKS
1	8	F	Bronchial asthma	Bicycle fall, brush burns and abrasions	0		
2	7	M	Angioneurotic edema	Stab wound from auto fender	0		
3	4	M	0	Stab wound of foot	0		
4	4½	M	Recurrent upper respiratory infections	Stab wound of hand	0		
5	14	M	Not known	Auto wound, brush burns and scratches from street	0		Unable to tolerate horse serum
6	4½	M	0	Rake cuts on face	Typical serum reaction with hives and generalized pains		Started within 24 hours; lasted 3 to 4 days
7	13	M	Bronchial asthma	Bicycle fall, brush burns and abrasions	Urticaria	8	Transient reaction for 3 days
8	8	M	0	Wrist cut from broken glass	Urticaria	4	Slight transient reaction
9	8	M	Allergic rhinitis	Crushing injury of hand; dirt ground in	Itching at site of injection	4	Transient reaction
10	2	F	0	Cut on forehead	0		
11	5	F	0	Stab wound with nail	0		
12	11	M	Pollinosis; bronchial asthma	Bicycle fall; cut arm	Urticaria	3	Lasted 5 days
13	18	M	Pollinosis; allergic rhinitis	Stab wound and lacerations of thigh; ski accident	Itching at site of injection	1	Transient reaction of 2 days' duration
14	5½	M	0	Cut on chin and knee	0		
15	4½	M	0	Stab wound from rake	0		
16	1½	M	Allergic rhinitis	Auto accident; extensive brush burns	Questionable		
17	6¼	M	Pollinosis	Cut over eye	0		
18	5¾	M	Eczema	Cut in leg from tin can	0		
19	13	M	0	Nail stab wound of foot	0		
20	4¼	M	Allergic rhinitis	Nail stab wound	0		

TABLE I—CONT'D

CASE NO.	AGE (YR.)	SEX	PERSONAL ALLERGY	NATURE OF INJURY	REACTION	DAYS	REMARKS
21	3½	M	Allergic rhinitis	Puncture wounds in soft palate from dirty stick	Mild urticaria	4	Transient reaction
22	12	M	Bronchial asthma	Bad firecracker injuries	0		
23	10	M	0	Cut on chin with dirty stick	Local swelling	1	Transient reaction
24	3½	M	0	Stab wound of upper lip	0		
25	3½	M	Bronchial asthma	Cut on knee with dirty stick	Urticaria	7	Transient reaction
26	4	F	0	Stab wound of face	0		
27	10	M	Pollinosis	Crushing injury of upper lip in coasting accident	Slight urticaria	2	Transient reaction
28	8	M	0	Stab wound of foot; nail	Hives, nausea	5	Reaction lasted only 1 day
29	8	M	Pollinosis	Stab wound of foot from nail	0		
30	3	M	0	Stab wound of hand from penholder	0		
31	10	F	0	Bicycle fall; multiple dirty abrasions	0		
32	2	M	0	Cut with unknown dirty object	Moderate hives	2	Lasted 2 days
33	14	M	0	Air-gun wound of wrist	0		
34	3	F	0	Auto accident; multiple severe dirty brush burns	0		
35	6	M	0	Firecracker wound of finger	Urticaria	7	Lasted 2 to 3 days
36	11	M	0	Stab wound of leg with rusty nail	0		
37	10	M	Perennial asthma, pollinosis	Firecracker wound of hand	0		
38	2½	M	Eczema; allergic rhinitis	Dog bite of face	Urticaria and angioneurotic edema	8	Lasted 3 days

telephone that he had had a slight generalized urticarial reaction starting on the fifth day and lasting for only twenty-four hours.

In three instances, Cases 1, 20, and 22, because of subsequent injuries weeks to months after the first injection of bovine tetanus antitoxin, it was necessary to repeat the injection. Disagreeable sequelae of any kind were not encountered.

DISCUSSION OF TABLE I

Eighty-two per cent of the twenty-eight patients listed in the table were males. This characteristic sex predominance is noted in all tables

of the incidence of tetanus or the use of prophylactic tetanus antitoxin. It is, of course, because of the greater outdoor activity and the more dangerous play and occupations of boys and men.

The personal histories of the patients in the table were known in all but Case 5. In the remaining thirty-seven children there was a personal incidence of allergic disease of approximately 49 per cent in the patients treated. Certainly a series of this type should be suitable for testing the safety of the parenteral injection of a foreign protein. I realize that many not well acquainted with pediatric allergy might question the frequent incidence of allergic rhinitis (other than pollinosis) in this series. However, this condition is one of the most common forms of allergy encountered in pediatric practice and represents an intermediate stage in the evolution of pollinosis or bronchial asthma.

Of the thirty-eight patients in the series, fifteen (39.5 per cent) experienced some reaction to the bovine antitoxin, varying from a questionable itching at the site of injection of the serum to a moderately severe serum sickness, which occurred in two instances. The incidence of serum reaction was equally divided between those patients with a personal history of allergy and those patients who had no personal history of allergy. In no case in this series was there a question of an immediate anaphylactic reaction to the bovine tetanus antitoxin.

Since the purpose of this presentation is to demonstrate the relative superiority of bovine serum over horse serum for the prophylaxis of tetanus, it would be desirable to compare this series with a series of horse serum injections as regards complications and sequelae. No immediate fatal anaphylactic reactions occurred with the bovine serum. Park[15] stated that this occurs about once in 70,000 cases with horse serum, and this incidence is probably less now than when his paper was published in 1921, as during this interval great improvements have been made in horse serum therapy. The series here reported is much too small to discuss from the standpoint of fatal anaphylaxis.

Weaver[16] reported that some type of serum reaction occurs in from 40 to 100 per cent of individuals to whom therapeutic sera are given. It is generally recognized that the incidence of serum reactions is proportional to the size of the dose, and that it also depends upon the method of administration. Tetanus antitoxin for prophylaxis is commonly given intramuscularly, so the factor which is important in my series is the size of the dose, which, as Gerlough[17] has demonstrated, is directly proportionate to the incidence of serum disease. The only published series of reactions following the prophylactic injection of horse serum for tetanus in amounts comparable to the bovine serum employed in my series is that of Lyall and Murdick.[18] These authors published the results of a series of 1,000 cases of all ages. Fifteen and two-tenths per cent of these developed generalized reactions (of which

only 0.2 per cent were alarming) as compared with two generalized reactions (Cases 6 and 28, neither of which was alarming), or 5.1 per cent, in my series. The protein content of the horse serum averaged 9 per cent; of the bovine serum, 7 per cent. My series, however, is so small that the above comparison with the series of Lyall and Murdick possesses only academic interest.

The question naturally arises as to whether or not there are any important objections to the use of bovine tetanus antitoxin. One objection which might be urged is that of cost. In New York state, horse serum tetanus antitoxin is provided without charge by the state. In states where tetanus antitoxin must be purchased, the average cost of 1,500 units of horse serum tetanus antitoxin is one dollar whereas that of the bovine preparation is four dollars. While cost might not be important with regard to prophylactic use of the serum, it might assume importance in treatment where large quantities must be used. In such cases it will be necessary for the physician to weigh carefully the advantages of the bovine serum against the disadvantages of the horse serum. The volume of the bovine tetanus antitoxin is about 3 c.c. or three times the volume of the horse serum for 1,500 units of horse serum tetanus antitoxin. This, however, cannot be regarded as a significant disadvantage.

Spaeth[5] stated, without specific reference to the literature: "Although the use of bovine antitoxin has been recommended. . . its limited immediate availability and the possibility of sensitivity to this material (despite negative cutaneous or ophthalmic reactions) make it an unsatisfactory substitute for general use." My personal experience shows the relative freedom from reactions following the use of bovine tetanus antitoxin and its invaluable aid in the treatment of children known to be sensitive to horse serum. Under present conditions bovine tetanus antitoxin has a world-wide distribution except for the limitations imposed by war. As prepared by the lyophil method, the serum will retain its potency at least five years, and probably indefinitely, even without adequate refrigeration. This being the case, there is no reason why the physician, if he is in a community where bovine tetanus antitoxin cannot be obtained on short notice, should not have in his office at all times a supply of the material at least sufficient for prophylaxis, if not for treatment.

Until bovine tetanus antitoxin is used much more extensively than at present, it cannot be stated with certainty that anaphylactic reactions and serum sickness are of less frequent occurrence with the bovine than with the equine serum. Nevertheless, the literature reviewed above and the short series here presented suggest that this is probably the case. It is clear from the evidence at hand that bovine tetanus antitoxin may be administered with safety to individuals who could not have been given horse serum without possibly fatal results. It is interesting

to speculate upon the reasons for this. Sensitivity to horse serum in our present population may be due in part to the sensitizing effect of the diphtheria toxin-antitoxin preparation used for the prophylaxis of diphtheria until the popular adoption by physicians of diphtheria toxoid. The sensitizing effect of the very minute amount of horse serum in the common toxin-antitoxin mixtures was first pointed out by Hooker[19] and emphasized by Tuft.[20]

It is generally recognized by allergists that allergic individuals tend to acquire sensitivity to some substances much more frequently than to others. To a certain extent this depends upon the degree of exposure of the individual to the substance, but to a larger extent it seems dependent upon something in the chemical or physical structure of the allergen. The term "sensitization potentiality" was suggested by the writer[13] to indicate that some substances are potentially capable of sensitizing the allergic individual more easily than other substances. Horse serum appears to be an allergen which has a particularly high sensitizing potentiality.

It is attractive to speculate that most individuals are immune to congenital or acquired sensitization to bovine serum, because the proteins of the serum are probably much the same, chemically and immunologically, as those of beef (although I have been able to find no references in the literature discussing this relationship), and through the ages, as man has been using beef as a food, he has acquired an immunity, except in rare instances, to the allergic effect of beef protein. Much the same may obtain in the case of the use of cow's milk as a human food. Of the four chief milk proteins, casein, lactalbumin, lactoglobulin, and an alcohol-soluble protein,[21] the globulin is known to be closely related to, if not identical with, the globulin of beef serum. I am inclined to believe, however, that the natural relative immunity of man to bovine serum is not due to inheritance for the reason that not only does the Darwinian theory state that acquired characteristics cannot be transmitted, but because cow's milk sensitivity is the most common form of allergy encountered in early infancy. Probably the infant normally undergoes a process of becoming immune to the ill effects of being fed cow's milk, and this immunity which usually persists for the remainder of life is responsible for the low "sensitization potentiality" of milk in older children and adults. Despite the great use of milk for nonspecific injection therapy a number of years ago and its continued use at present, though to a much less extent, I have been able to find the records of but three cases of individuals who were sensitized to milk by the injection of milk protein.[22-24] These observations are significant in connection with the statement of Wells and Osborne,[21] that animals fed with milk are not good subjects upon which to try anaphylaxis experiments with milk, as they may have acquired more or less of an immunity. I have had no experience with what

might happen if the milk-sensitive or beef-sensitive individual were given bovine serum. I had one 4-month-old infant with eczema who gave strongly positive transfer tests to cow's milk, beef muscle, and bovine tetanus antitoxin.

Although serum disease may result from the injection of human serum,[25] and despite the presence of a species nonspecific antigen factor in mammalian serum,[26] an ideal tetanus antitoxin could probably be made available by the use of human serum from donors previously immunized by tetanus toxoid, as suggested by Hyland.[27] With the extensive development of serum centers throughout the country, Hyland's suggestion is very practical. By this method a tetanus antitoxin could be developed whose "sensitization potentiality" would probably be very close to zero. The work on despeciated horse serum reported by Coghill, Fell, Creighton, and Brown[28] also promises to be of great value in the therapeutic use of horse serum.

CONCLUSIONS

Bovine tetanus antitoxin has been used with safety in selected cases of individuals known to give severe anaphylactic reactions to horse serum.

It is suggested that bovine tetanus antitoxin has a lower "sensitization potentiality" than the equine preparation and should be used as a substitute for the equine serum in all cases, except as below noted.

The only indication for the use of equine tetanus antitoxin occurs in those cases where the individual is known to be, or can be demonstrated to be, sensitive to the bovine but not to the equine preparation. Thus far no such cases have been reported.

Individuals undergoing an allergic study should be tested routinely with equine and bovine sera. This should be done by the scratch method only to avoid sensitizing the individual to the serum. If the individual is sensitive to both sera, he should be immunized by means of tetanus toxoid, the precautions suggested by Cooke and his co-workers[29] being carefully observed.

The production of human antisera as well as the use of other measures to increase the safety of serotherapy is greatly to be encouraged.

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Critical Review

DISORDERS OF INTERNAL GLAND SECRETION IN CHILDREN

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AFTER a survey of the literature it was decided to restrict the present review to a discussion of the thyroid and the adrenal cortex. From the point of view of the pediatrician, the actions of these glands can be more accurately defined than was possible in the past, and the way is being opened to newer conceptions upon which to base treatment.

THYROID

Some years ago it was believed that the physiology and actions of the thyroid gland were completely understood. Recently, studies of its action by means of new methods have opened the field anew and have shown that there is still much to be learned. The following studies have done much to define more clearly many phases of thyroid activity which in the past were not suspected.

Salter[1] recently has summarized in monograph form the present knowledge of the relation between iodine metabolism and thyroid function. Certain of the organic iodine-containing constituents of the blood plasma are of particular interest. The plasma "P" iodine concentration is consistently low in hypothyroid patients. When thyroxine is administered to a myxedematous patient, the plasma "P" iodine level rises; similarly it is found also to be elevated in patients with spontaneous hyperthyroidism. These observations suggest that determinations of the concentration of circulating plasma "P" iodine may give more exact physiologic information concerning thyroid gland activity than the indirect procedures, such as the basal metabolic rate, etc., employed at present. Unfortunately, the methods for making blood iodine measurements are intricate and require more technical skill than is available generally for routine clinical use.

Wilkins, Fleischmann, and co-workers[2-4] have reappraised the usefulness of the basal metabolism determination, the serum cholesterol value, and the creatine excretion and tolerance test as aids in the diagnosis of childhood hypothyroidism. They feel that the basal metabolic rate is not satisfactory because of the difficulties encountered in determining the oxygen consumption and in interpreting the values obtained. These reported difficulties are surprising in view of the large number of successful determinations of the basal metabolism which have been made by others on both infants and children. In hypothyroid children who had not received thyroid extract, the creatine tolerance and serum cholesterol value were frequently within normal limits. On the other hand,

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when thyroid was administered to such children, there was a tendency for the serum cholesterol concentration to fall more in hypothyroid than in normal subjects. When thyroid medication was discontinued, the serum cholesterol value of hypothyroid children rose to abnormally high levels, whereas it remained within normal limits in normal children similarly treated. Goodkind and Higgins[5] also have recently reported new data which show that determinations of the basal metabolic rate and blood-cholesterol level following the withdrawal of thyroid therapy are of diagnostic significance in doubtful cases of treated cretinism. The point deserves emphasis that a normal serum cholesterol level does not rule out hypothyroidism in an infant or child who has not received thyroid medication. Indeed, it has been our experience that a low cholesterol value is as common as an elevated value in such individuals. Radwin and his associates[6] suggest that the total blood lipids are more consistently elevated than the blood cholesterol in childhood hypothyroidism. This possibility merits further study.

The serum phosphatase measurement, expressed in Bodansky units, appears to provide a simple and reliable index of thyroid deficiency during infancy and childhood.[7] The serum phosphatase level was found to be abnormally low (less than 4.5 Bodansky units) in hypothyroid children who had not received thyroid extract. It was restored to normal levels by the administration of adequate amounts of thyroid hormone. With the possible exception of certain cases of idiopathic dwarfism, a lowering of the serum phosphatase level was not observed in children suffering from diseases which easily might be confused with hypothyroidism.

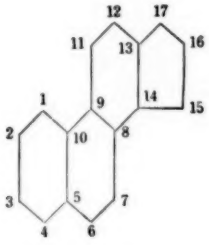
The response of children to the administration of anterior pituitary thyrotropic hormone has been studied by Wilkins and Fleischmann.[8] When administered to normal children, it caused an increase in the excretion of creatine in the urine. This was interpreted in the light of earlier studies to indicate a response of the child's thyroid gland to the administered pituitary hormone. Nine hypothyroid children showed no response to the thyrotropic hormone, although they did excrete more creatine when thyroid extract was given. These interesting observations open new avenues of approach to the problem of determining the nature of hypothyroidism in children. When correlated with the studies of others[9, 10, 11] who have shown that pituitary thyrotropic hormone may be excreted in excessive amounts in the urine of hypothyroid patients, these observations suggest that the thyroid deficiency is due to absence, atrophy, or lack of responsiveness of the thyroid gland rather than to pituitary deficiency. In view of these findings it is not surprising that anterior pituitary therapy was found to be ineffective in the treatment of juvenile hypothyroidism.[12]

The question of the cause of mental deficiency in cretins has been reopened by Goodkind and Higgins.[5] They find that contrary to the general belief, "the age at which the diagnosis was made and treatment started was by no means the deciding factor in the ultimate mental capacity." Because the ultimate mental development of adequately treated cretins was not related strictly to physical growth, they feel that the cerebral deficiency probably can be regarded as congenital and from the same cause as the thyroid damage. On the other hand, the observations of Dorff[13] and Elmer and Schepps[14] suggest that the mental deficiency is due to lack of thyroid during the latter part of fetal develop-

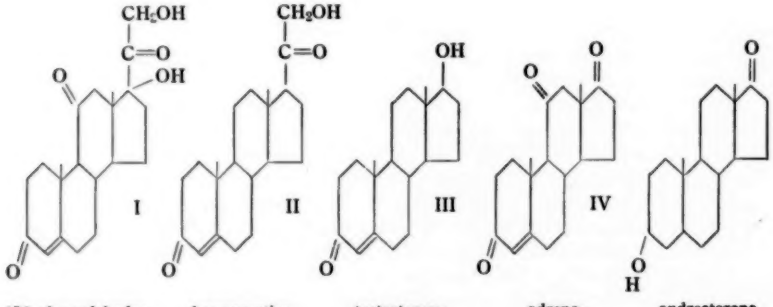
ment. The latter authors have presented evidence which shows that thyroxine is secreted in fetal life and which suggests that the fetus is not dependent upon the mother for thyroid hormone. These authors postulate a disturbance in the germ plasm for the anlage of the thyroid, particularly in cases of sporadic cretinism. This disturbance results in fetal athyreosis. It is hard to accept this postulate since it does not explain why the majority of sporadic cretins are normal at birth, apparently being born with sufficient thyroid. In most instances the sporadic cretin shows no signs of hypothyroidism until some time in the third month, in contrast to the true congenital cretin who has all the signs of hypothyroidism at birth. More evidence is needed before this question can be settled. All are agreed, however, that the optimal prognosis for any given cretin depends upon early recognition of the thyroid deficiency and prompt and adequate therapy. There is nothing to suggest that excessive doses of thyroid extract will improve the prognosis.

ADRENAL CORTEX

Knowledge of the functions of the adrenal cortex has been extended greatly in recent years. This advance has been facilitated by the isolation of approximately twenty different hormones in crystalline form from adrenal cortex tissue by Reichstein and his associates in Europe and Kendall, Wintersteiner, and others in this country. These are called steroid hormones because, like cholesterol, they all have a nucleus of carbon atoms arranged in a system of four rings as shown below:



Each carbon is numbered for convenience in describing the individual variations which characterize each substance.



17-hydroxydehydrocorticosterone | desoxycorticosterone | testosterone | adrenosterone | androsterone

From the physiologic point of view, the chemistry of these hormones is interesting because there appears to be a relationship between structure and biologic activity.[15-17] Biologically active adrenal cortical steroids have a ketone at carbon 3 with an unsaturated carbon atom linkage adjacent to it. They also have a side chain containing an hydroxyl and another ketone group attached to carbon 17 (I and II). The activity of these compounds is apparently modified by the presence or absence of an oxygen atom at carbon 11. Thus for example 17-hydroxydehydrocorticosterone (I), which has an oxygen at carbon 11, predominantly acts upon the carbohydrate metabolism, whereas desoxycorticosterone (II), which lacks an oxygen at carbon 11, predominantly influences water and electrolyte metabolism. The diagram shows that these two adrenal hormones are quite similar to testosterone (III), which is a potent masculinizing hormone (androgen) obtained from testis tissue and adrenosterone (IV), which is an adrenal androgen and androsterone (V), and which is one of the urinary androgens.

The relationship of adrenal cortical hormones to carbohydrate metabolism[18] as it is now understood may be summarized as follows: Certain hormones of the adrenal cortex (such as 17-hydroxydehydrocorticosterone, see above) promote the formation of carbohydrate from protein. If there is a deficiency of these hormones, this gluconeogenesis is depressed; when there is an excess of the hormones, gluconeogenesis is accelerated. As a consequence of accelerated gluconeogenesis, carbohydrate accumulates as glycogen in the liver and muscles and as glucose in body tissues and fluids at the expense of tissue proteins. Thus there may be hyperglycemia and glycosuria. In this sense these particular hormones are diabetogenic. On the other hand, a depression in the rate of gluconeogenesis may not be observed unless the animal is fasted. Under these circumstances the carbohydrate stores become rapidly depleted, and hypoglycemia develops because of the depressed gluconeogenesis. An increase in the utilization of available carbohydrate in adrenal cortical deficiency hastens this depletion.[19]

These effects of adrenal cortical hormones on carbohydrate metabolism may be observed clinically. Hypoglycemia has long been encountered in patients with cortico-adrenal insufficiency (Addison's disease).[20] Thorn and co-workers[17] suggest that this hypoglycemia may be elicited by the application of a standard intravenous glucose tolerance test. A striking hypoglycemia develops in two to three hours. Fraser, Albright, and Smith[21] have shown that patients with cortico-adrenal insufficiency are also "hypoglycemia unresponsive." This was demonstrated by a standardized intravenous insulin tolerance test. Following the administration of insulin, the blood sugar level of patients with cortico-adrenal insufficiency remains low much longer than in the normal individual. On the other hand, "hyperglycemia unresponsiveness" can be demonstrated in patients with Cushing's disease by the oral glucose test and the combined glucose-insulin tolerance test. This latter finding leads them to believe that Cushing's disease is due to an overproduction of glyconeogenetic hormones by the adrenal cortex.[22] Testosterone apparently counteracts this tendency toward excessive conversion of tissue proteins to carbohydrate in Cushing's disease and is therefore a promising therapeutic agent in that disease.

Cortico-adrenal insufficiency also results in the more familiar disturbances in water and electrolyte metabolism.[23-28] These disturbances are explained at least in part by alterations in renal function. The kidneys lose their ability (a) to excrete catabolites, such as potassium, which

occur in low concentrations in the plasma and which are normally excreted by a process of concentrating them in the urine; and (b) to stop excreting sodium and chloride when the plasma concentrations of these electrolytes fall below normal levels.

Desoxycorticosterone and its esters are particularly effective in correcting these water and electrolyte effects of cortico-adrenal insufficiency. If they are administered in excessive doses, they are capable of causing retention of sodium, chloride, and water and excessive excretion of potassium. As a consequence, edema, hypertension, and cardiac decompensation may develop as well as temporary muscular paralysis due apparently to low serum potassium. Details of correct dosage are given by authors listed in the bibliography.[29-32]

The role of the adrenal cortex in the production of sex hormones is not usually evident clinically except when the sex hormones are produced in excessive amounts such as are found in hyperplasia or carcinoma of cortico-adrenal cells. In children the excess hormones cause the familiar evidences of pseudosexual precocity. This is usually masculine in type,[33] probably because the excess hormones are usually predominantly androgenic. Instances of feminine pseudosexual precocity due to cortico-adrenal tumors also have been observed, however.[34]

Callow and others[35-42] and Butler and others[43-46] have presented evidence which indicates that some of the cortico-adrenal and testicular hormones are converted by the body into neutral 17-ketosteroids which are excreted in the urine. The present evidence indicates that of the urinary 17-ketosteroids, the alpha-hydroxy compounds, such as androsterone, and 3-a-hydroxyetiocholanone-17, arise from substances elaborated in both the testes and adrenal cortex; on the other hand, the beta-hydroxy compounds, such as dehydroisoandrosterone and isoandrosterone, are apparently derived solely from substances produced by the adrenal cortex. The beta hydroxyketosteroids normally comprise approximately 10 per cent of the total 17-ketosteroids.[46]

The rate of excretion of 17-ketosteroids is normally low in children up to approximately 10 years of age.[46-48] The output increases and rapidly approaches adult levels at about the time of puberty. Because the hormones of the testes, but not of the ovaries, give rise to urinary 17-ketosteroids, the values in adult males average about 40 per cent higher than in the adult females.[49]

In patients with cortico-adrenal hyperplasia there is a moderate increase above normal limits in the excretion of both alpha and beta 17-ketosteroids,[46] whereas a marked increase particularly in the excretion of beta 17-ketosteroids is observed in patients with cortico-adrenal carcinoma.[40, 41, 43, 46, 49, 50, 51] Because comparable increases in 17-ketosteroid excretion are not observed in patients with simple hirsutism, Cushing's syndrome without adrenal tumor, ovarian arrhenoblastoma, or sexual precocity due to intracranial lesions,[46, 49] the measurement of alpha and beta 17-ketosteroids in the urine provides useful information in the diagnosis of hyperplasia and carcinoma of cortico-adrenal cells.

On the other hand, a lowering in the 17-ketosteroid excretion rate may be of significance provided the patient is at least 10 or more years of age. In children younger than 10 years the 17-ketosteroid excretion rate is normally so low that hypoexcretion cannot easily be detected with accuracy. In adults a lowered excretion rate is observed in patients with the classical form of Addison's disease.[42, 49] This is explained by hypo-

function of the adrenal cortex. As would be expected, the values are usually lower in female patients with Addison's disease than in male patients. A marked lowering is encountered in patients with hypofunction of the anterior pituitary (panhypopituitarism).[42, 49] This is explained by the fact that the activity of the gonadal and cortico-adrenal cells is dependent upon the stimulus provided by pituitary gonadotropic (gonad stimulating) and adrenalotropic hormones. Castration results in a partial lowering of 17-ketosteroid excretion, particularly in the male.[42, 49] Hypothyroidism also causes a diminution in the rate of 17-ketosteroid excretion.[49]

A few cases of cortico-adrenal insufficiency (Addison's disease) in children[52, 53] and a number of instances of the Waterhouse-Frederickson syndrome have been recorded in the recent literature. In addition, reports have been published on two male infants[54, 55] with masculine pseudosexual precocity who were also suffering from a disturbance in water and electrolyte metabolism indistinguishable from that observed in the classical form of Addison's disease. One of these infants[55] died and at post-mortem hyperplasia of the so-called androgenic tissue of the adrenal cortex was found. In retrospect it was evident that this infant had had a great craving for salt and that by spontaneously increasing his salt intake he had kept himself alive for two years.[55] The other patient[56] almost died from lack of salt on several occasions before the diagnosis of cortico-adrenal insufficiency was made.

The symptoms referable to the deficiency of vital adrenocortical hormones in these infants were essentially the same as the symptoms observed in any infant with an acute nutritional disturbance accompanied by severe dehydration and circulatory collapse. The diagnosis was suggested by brownish pigmentation of the genitalia and nipples, a low serum sodium, and an elevated serum potassium level. In addition, it was demonstrated that the urinary excretion of estrogens and of 17-ketosteroids[54] was abnormally elevated. The latter finding is in contrast to the abnormally low 17-ketosteroid excretion usually observed in adult patients with the classical form of Addison's disease[42, 49] but is consistent with the elevated excretion rate noted in patients with cortico-adrenal hyperplasia.[46, 49] The present evidence thus suggests that the adrenal cortex of these infants was producing an excess of sex hormones but was not producing "life maintaining" hormones in sufficient quantities.

The details of treatment of adrenocortical deficiency have been adequately reviewed by others[57, 58] and therefore will not be discussed here.

Our point of view concerning the other glands and glandular preparations discussed in the preceding reviews has not changed materially.

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Proceedings

TENTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

MEMPHIS, TENN.—NOV. 18-20, 1940

Round Table Discussion on Appraisal of Quarantine Requirements

Chairman: Archibald L. Hoyne, M.D., Chicago, Ill.

Associates: Philip M. Stimson, M.D., New York, N. Y.
Franklin H. Top,* M.D., M.P.H., Detroit, Mich.

Secretary: Sterling H. Ashmun, M.D., Dayton, Ohio

DR. TOP.—The title of the symposium, I presume, includes a consideration of both isolation and quarantine measures. There has been considerable confusion as to the meaning of these two terms, and for the purpose of this paper, and I suppose for the symposium, the definitions given in the monograph on the "Control of Communicable Diseases," published by the United States Public Health Service, will be used.[1] Quarantine denotes the restriction of healthy contacts to premises, while isolation is taken to mean the separation of the known sick or carriers from others in home or hospital.

Scarlet Fever

In a consideration of all communicable diseases, the control of scarlet fever probably has been one of the most unsatisfactory as compared to that for diphtheria and typhoid fever, diseases for which definite regulations have been of distinct value. It is not surprising that this should be the case. From a definite disease entity, due to a single strain of hemolytic streptococcus, as considered by the Dicks,[2] the disease has become a syndrome; namely, one of a number of hemolytic streptococcus infections caused by beta hemolytic streptococci of the Lancefield A group,[3] of which there are twenty-seven strains, as classified by Griffith.[4] Scarlet fever differs from other streptococcal infections in that the streptococci responsible produce an erythrogenic or rash-producing toxin. All of the strains isolated from scarlet fever patients produce the same kind of toxin, and in this they are similar. There is, however, little cross protection between strains as far as bacterial immunity is concerned.

In the past there has been wide variation in quarantine and isolation practices among the states of the Union and in foreign countries. Twenty years ago isolation periods exceeding six weeks were not uncommon, but the usual period of isolation now is twenty-eight days. In general, American practice in the past ten years has inclined toward shortening of isolation periods, although Darling and Gordon[5] found extreme variation in practice among urban communities in the United States in 1932. In a few communities a period as short as two weeks has been utilized with no apparent increase in the incidence of the disease. In an occasional locality isolation

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for scarlet fever has been abolished. Holst's[6] experience in Bergen and Trondheim is evidence that isolation and quarantine procedures have made no practical difference in the incidence of scarlet fever in these two cities, one of which carried out strict quarantine and isolation restrictions, whereas the other had no specific regulations whatsoever. Some communities in England eliminated restrictions without untoward effects. To emphasize further the apparent inadequacy of control measures for scarlet fever, numerous communities in England have hospitalized more than 90 per cent of their scarlet fever cases with the hope that the incidence of the disease would be lowered. During a period of over twenty-five years, Woods[7] found no material drop in the incidence of scarlet fever in these communities or in England at large. With these facts in mind, we present the experience of the city of Detroit for the years 1929 through 1938.

Changes in the Isolation Time of Scarlet Fever in Detroit.—The isolation time of scarlet fever has been materially changed in Detroit during the last ten years. In Table I it will be noted that the isolation period was four weeks for all ages

TABLE I

MINIMUM ISOLATION TIME FOR UNCOMPLICATED SCARLET FEVER IN DETROIT

TIME	ISOLATED 4 WK.	ISOLATED 3 WK.	ISOLATED 2 WK.
Jan. 1, 1929, to June 30, 1930	All ages		
July 1, 1930, to June 30, 1931	Under 20 yr.	20 yr. and over	
July 1, 1931, to Nov. 9, 1931		All ages	
Nov. 10, 1931, to June 30, 1932	Under 15 yr.	15 yr. and over	
July 1, 1932, to Dec. 31, 1932		Under 15 yr.	15 yr. and over
Jan. 1, 1933, to June 30, 1933	Under 15 yr.	15 yr. and over	
July 1, 1933, to Dec. 31, 1933		Under 15 yr.	15 yr. and over
1934 to 1940		Under 15 yr.	15 yr. and over

prior to July 1, 1930. During the next year patients under 20 years of age were isolated four weeks and those over this age, for a period of three weeks. In all instances noted in Table I the periods are minimum periods for uncomplicated cases of scarlet fever. From July 1, 1931, to Nov. 9, 1931, a period of low scarlet fever incidence, patients at all ages were isolated for a period of three weeks. From Nov. 10 through June 30, 1932, persons under the age of 15 years were isolated four weeks, and those 15 years of age and over, for a period of three weeks. Although the period of isolation remained the same in 1931 and 1932 as compared with 1930 and 1931, the age limit dropped five years, which was a definite concession. From July 1, 1932, to Dec. 31, 1932, patients under 15 years of age were isolated for three weeks, and those 15 years of age and over, for a period of two weeks. During the next six months, when the incidence of scarlet fever was greater, patients under 15 years of age were again isolated for four weeks, and those 15 years and over, for a period of three weeks. From July 1, 1933, through Dec. 31, 1933, the regulations obtaining in the same period of 1932 were in force. A study made by Gordon

and Badger[8] demonstrated the feasibility of lowering the isolation period throughout the year for both the "under 15" and "over 15" age groups. Consequently, in 1934, and continuing to the present, patients under 15 years of age have been and are isolated for a period of three weeks, while those 15 years and over are isolated two weeks. The only concession made for the winter and spring season is an additional seven days of modified isolation for patients 15 years of age and under, during which time they are barred from school but are granted freedom in the home.

TABLE II

CASES, INFECTING CASES, AND INFECTING CASE RATES BY AGE GROUPS

YEAR	NO. OF PATIENTS			INFECTING CASES			INFECTING CASE RATES*		
	0-14 YR.	15+ YR.	ALL AGES	0-14 YR.	15+ YR.	ALL AGES	0-14 YR.	15+ YR.	ALL AGES
1929	2,220	788	3,008	110	4	114	5	0.5	3.8
1930	1,521	397	1,918	61	3	64	4	0.8	3.3
1931	1,753	400	2,153	59	3	62	3.4	0.8	2.9
1932	2,599	454	3,053	93	6	99	3.6	1.3	3.2
1933	1,766	297	2,063	68	2	70	3.9	0.7	3.4

Data from the Herman Kiefer Hospital, 1929 to 1933.

*Infecting case rate is the percentage of patients released from the hospital known to be responsible for familial scarlatinal infections within thirty days after release.

Table II, which is taken from Gordon and Badger's article, shows hospital cases, infecting cases, and infecting case rates by age groups for the years 1929 through 1933 and covers the experience with isolation procedures prior to 1934 noted in Table I. By an infecting case is meant a patient who is released from hospital or home at the end of a prescribed period of isolation and known to have been responsible for a familial scarlatinal infection within thirty days after release. An infecting case rate is the percentage of patients released from hospital or home known to be responsible for familial scarlatinal infections within thirty days after release. It is obvious from an inspection of Table II that the infecting case rates by age did not increase significantly as the result of less stringent restrictions placed on scarlet fever patients after 1929. It is also equally clear that persons 15 years of age and over became infecting cases far less frequently than did those under the age of 15. With this evidence in mind, in 1934 the health authorities in Detroit adopted the shortened isolation periods now in effect in Detroit.

The season of the year influences the infecting case rate, as is evident in Table III. In both Tables II and III, 1929 is to be taken as a year of average experience for a period during which the isolation time for all ages was twenty-eight days. In the latter table it will be noted that in general the infecting case rates are highest during the first three and the last three months of any calendar year. For this reason an additional seven days of modified isolation is required for patients under 15 years of age during the winter months.

Succeeding tables show Detroit's experience with the shortened isolation period for the years 1934 to 1938. Compared to Table II, of which it is a pattern, Table IV shows lower infecting case rates each succeeding year with but few exceptions; and this also obtains for Table V, which is similar to Table III. It must be borne in mind that scarlet fever has become progressively milder in the last ten years, and this fact may be solely responsible for the lowered infecting case rates. However, under present conditions there is no necessity for maintaining extended periods of isolation as long as the disease is so mild.

Throughout the years that isolation studies were carried on, it was evident that fewer infecting cases resulted following isolation of patients at home than when cases were isolated in hospital. This is amply demonstrated in Table VI, which shows the number of infecting cases and the infecting case rates by age groups

TABLE III

CASES, INFECTING CASES, AND INFECTING CASE RATES BY SEASON AND YEAR

YEAR	NO. OF PATIENTS					INFECTING CASES					INFECTING CASE RATES				
	JAN. TO MAR.	APRIL TO JUNE	JULY TO SEPT.	OCT. TO DEC.	TOTALS	JAN. TO MAR.	APRIL TO JUNE	JULY TO SEPT.	OCT. TO DEC.	TOTALS	JAN. TO MAR.	APRIL TO JUNE	JULY TO SEPT.	OCT. TO DEC.	TOTALS
1929	847	1,290	386	485	3,008	33	39	8	34	114	3.9	3	2.1	7	3.8
1930	708	716	237	257	1,918	27	28	2	7	64	3.8	3.9	0.8	2.7	3.3
1931	545	968	412	228	2,153	27	21	9	5	62	5	2.2	2.2	2.2	2.9
1932	863	1,451	462	277	3,053	37	41	8	13	99	4.3	2.8	1.7	4.7	3.2
1933	699	914	166	284	2,063	26	24	5	15	70	3.7	2.6	3	5.3	3.4

Data from the Herman Kiefer Hospital, 1929 to 1933.

TABLE IV

HOSPITAL CASES, INFECTING CASES, AND INFECTING CASE RATES BY AGE GROUPS

YEAR	NO. OF PATIENTS			INFECTING CASES				INFECTING CASE RATES		
	0-14 YR.	15+ YR.	ALL AGES	0-14 YR.	15+ YR.	UNDETERMINED	ALL AGES	0-14 YR.	15+ YR.	ALL AGES
1934	1,555	271	1,826	50	5	3	58	3.2	1.8	3.2
1935	1,453	251	1,704	45	4	6	55	3.1	1.6	3.2
1936	1,820	279	2,099	50	4	2	56	2.7	1.4	2.7
1937	2,981	454	3,435	93	5	5	103	3.1	1.3	3
1938	1,815	235	2,050	48	1	6	55	2.6	0.4	2.7

Data from the Herman Kiefer Hospital, 1934 to 1938.

following the home isolation of scarlet fever cases. The difference in infecting case rates between home and hospital isolated cases can be attributed to the greater proportion of complications among hospital patients, a clinical observation of many years and one confirmed as due to reinfection by the work of Gunn and Griffith[9] and that of Allison and Brown.[10] The patient who comes to a hospital with scarlet fever resulting from one strain of hemolytic streptococcus may develop complications as the result of a different strain picked up while in the hospital and on dismissal may carry the new strain into the home. There is a hazard in hospital isolation which should lead to the home care of scarlet fever for those mildly infected, but patients seriously ill are better cared for in hospital.

A return case is a secondary case of scarlet fever occurring within thirty days of release of a primary case from home or hospital. Return case rates have been used as a yardstick for determining the value of isolation procedures, but it does not give a true picture of the problem because several return cases may have been caused by one infecting case. A true perspective can be gained only by knowing the actual number of released cases which result in one or more return cases. The occurrence of one or more return cases is a fortuitous circumstance, dependent upon the number of susceptible persons an infecting case may come into contact with. For purposes of comparison, Tables VII and VIII give the total number of infecting and return cases for the years 1934 through 1938, with case rates and the number of return cases per infecting case for both hospital and home experience. In some instances the difference between infecting and return cases is small, and the difference in the rates is likewise insignificant. During these years the ratio of infecting cases to return cases was not greater than 1 to 1.4 for hospital infecting cases and 1 to 1.3 for home infecting cases. It is obvious that there is variation from year to year in both the infecting and return case rates. This is explained by the incidence of the disease in the city at large. As a general rule, during years in which the disease assumes epidemic proportions, the infecting and return case rates are higher. The years 1936 and 1938 showed a relatively low scarlet fever incidence, whereas in 1937 almost three times as many cases were reported in the city as during 1936 and 1938.

Changes in Quarantine Regulations for Scarlet Fever in Detroit.—In former years exposed adults and children living in the house with the patient were restricted to the premises until the patient in the home was released from isolation. The exception to this was the removal of exposed adults or children to the homes of relatives or friends where they were quarantined for a period of seven days from the time of last possible contact with the patient. In the spring of 1939 the quarantine regulations relating to adults were modified so that the breadwinner might remain in the home if facilities for home isolation of the patient were satisfactory and if the breadwinner was not a food-handler and did not come into contact with children. This regulation has been in effect for too short a time to warrant a definite

TABLE V

CASES, INFECTING CASES, AND INFECTING CASE RATES BY SEASON AND YEAR

YEAR	NO. OF PATIENTS					INFECTING CASES					INFECTING CASE RATES				
	JAN. TO MAR.	APRIL TO JUNE	JULY TO SEPT.	OCT. TO DEC.	TOTALS	JAN. TO MAR.	APRIL TO JUNE	JULY TO SEPT.	OCT. TO DEC.	TOTALS	JAN. TO MAR.	APRIL TO JUNE	JULY TO SEPT.	OCT. TO DEC.	TOTALS
1934	716	575	109	426	1,826	31	7	2	18	58	4.3	1.2	1.8	4.2	3.2
1935	781	574	65	284	1,704	34	7	2	12	55	4.4	1.2	3.1	4.2	3.2
1936	626	777	158	538	2,099	21	15	4	16	56	3.4	1.9	2.5	3	2.7
1937	1,615	1,172	220	428	3,435	65	16	2	20	103	4.0	1.4	0.9	4.7	3
1938	728	647	158	517	2,050	15	20	2	18	55	2.1	3.1	1.3	3.5	2.7

Data from the Herman Kiefer Hospital, 1934 to 1938.

TABLE VI

HOME CASES, INFECTING CASES, AND INFECTING CASE RATES BY AGE GROUPS

YEAR	NO. OF PATIENTS			INFECTING CASES				INFECTING CASE RATES		
	0-14 YR.	15+ YR.	ALL AGES	0-14 YR.	15+ YR.	UNDETERMINED	ALL AGES	0-14 YR.	15+ YR.	ALL AGES
1934	2,346	226	2,572	28	1	3	32	1.2	0.4	1.2
1935	1,927	161	2,088	15	2	3	20	0.8	1.2	1
1936	2,473	166	2,639	12	2	1	15	0.5	1.2	0.6
1937	6,699	557	7,256	63	4	7	74	0.9	0.7	1
1938	2,821	178	2,999	18	-	2	20	0.6	-	0.7

Data from the city of Detroit, 1934 to 1938.

TABLE VII

HOSPITAL CASES, INFECTING CASES, RETURN CASES, INFECTING AND RETURN CASE RATES AND RETURN CASES PER INFECTING CASE BY YEAR

YEAR	TOTAL CASES	INFECTING CASES	RETURN CASES	CASE RATES		RETURN CASES PER INFECTING CASE
				INFECTING	RETURN	
1934	1,826	58	75	3.2	4.1	1.3
1935	1,704	55	74	3.1	4.3	1.3
1936	2,099	56	75	2.7	3.6	1.3
1937	3,435	103	141	3.1	4.1	1.4
1938	2,050	55	66	2.6	3.2	1.2

Data from the Herman Kiefer Hospital, 1934 to 1938.

TABLE VIII

HOME CASES, INFECTING CASES, RETURN CASES, INFECTING AND RETURN CASE RATES AND RETURN CASES PER INFECTING CASE BY YEAR

YEAR	TOTAL CASES	INFECTING CASES	RETURN CASES	CASE RATES		RETURN CASES PER INFECTING CASE
				INFECTING	RETURN	
1934	2,572	32	34	1.2	1.3	1.1
1935	2,088	20	25	1	1.2	1.3
1936	2,639	15	17	0.5	0.6	1.1
1937	7,256	74	79	1.0	1.1	1.1
1938	2,999	20	20	0.7	0.7	1

Data from the city of Detroit, 1934 to 1938.

statement as to its effect upon the incidence of scarlet fever. To date, no case has been directly traceable to a breadwinner who received a permit to live in the home where a patient was isolated with scarlet fever.

Isolation and Quarantine Regulations for Other Communicable Diseases

In general, the recommendations found in the monograph "Control of Communicable Diseases," cited above, are ample and justified. Perhaps a few minor changes could be instituted without risk to the public health. A year ago contacts to patients with meningococcus meningitis were required to remain in quarantine for a period of fourteen days. At the present time they are held for a period of seven days. The disease has become so infrequent in Detroit that in 1939 but six cases were reported. In the light of this low incidence such a change seems justified. When changes in regulations for the control of communicable diseases in the state of Michigan were considered in the spring of 1939, a modification was advocated

which would have altered the quarantine period for contacts to poliomyelitis cases from fourteen to seven days. The change was not made. We believe that the shortened period would not have materially influenced the incidence of this disease, particularly during a period of low incidence.

The minor communicable diseases such as chicken pox, German measles, mumps, and even measles are so poorly reported and are in general so mild that isolation or quarantine regulations cannot very well be enforced successfully, although it is advantageous to the public and patient to do so. However, it seems to us that in the instance of measles it is highly desirable that infants and small children below the age of 4 years be isolated from others, including members of the family, relatives, and friends, except, of course, the nurse or attendant. The child in this age group is less of a menace to the public health than the public is to the child's health. There is great danger that others on visiting the child may be the source of pathogenic organisms, particularly streptococci and pneumococci, which may result in pneumonia or other severe complications. The same holds true for a disease like whooping cough, a disease of relatively long duration, and one in which pneumonia is common under the age of 4 years.

New diagnostic methods, better sanitary conditions in the home, and improved economic status of the public at large have contributed to a lowered incidence of communicable disease, to ignore the influence of immunization procedures. Many of the restrictions now placed upon patients with communicable disease appear to be as lenient as possible, consistent with the protection of the public health. For some diseases, such as scarlet fever, it may seem feasible to shorten further the isolation period. When the patient is taken into consideration, however, a minimum of twenty-one days seems desirable for children under the age of 15 years. Complications in scarlet fever are most commonly encountered in the latter part of the second week and during the third week of the disease. If the isolation period is shortened, the patient will be allowed liberties which will enhance the possibility of contracting a complication which may prove serious. In mumps, a minor communicable disease, isolation restrictions are not strictly enforced, and yet a definite period of isolation might result in some patients remaining bedfast until all evidence of swelling of affected glands had disappeared. Isolation restrictions of the future should direct greater emphasis on protection of the patient and, by so doing, secondarily protect the public health. It should not be forgotten, however, that quarantine and isolation restrictions of necessity must be modified periodically by changes in the prevalence of communicable diseases.

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DR. STIMSON.—It has been shown recently that if there is a case of poliomyelitis in a locality there will be three or more children also with upper respiratory or alimentary infections but nothing else to indicate poliomyelitis. If you isolate the case with paralysis while the three or more walk about, you do not accomplish anything; but if you isolate everyone who is sick, you might accomplish something. However, it is pretty difficult to isolate all the others.

At the Willard Parker Hospital in New York this last summer we had fifteen cases of poliomyelitis, only one of them being of the bulbar type. Many of the bulbar cases are harmed by the respirator. What happens is that the mucus in the throat is sucked down into the chest, and the patient cannot cough it up and spit it out. Landon and Ellis summarized our figures of 1931 for spinal cases; at that time it happened that we had about as many patients who needed a respirator but could not have one because it was not available, as patients who were put in a respirator. The two groups are fairly comparable. There were about seventy in each group. The mortality was about equal in the two groups, but a year later, five times as many of the patients who had been in the respirator had died. We have learned now to handle the respirator more carefully, and we use 10 cm. or less of water pressure. This is very important.

There are five general types of respiratory involvement in poliomyelitis: First, a simple intercostal involvement, in which a respirator helps; second, simple diaphragmatic involvement, in which a respirator helps; third, a mixture of the first and second types in which a respirator helps; fourth, involvement of the respiratory center and toxic involvement of the respiratory center usually accompanied by bulbar involvement, in which the respirator does not help at all. In the fifth type, no direct paralysis of the muscles of respiration and no direct involvement in the respiratory center occur, but there are bulbar manifestations so that the patient cannot cough or swallow. The mucus accumulates in the throat and gets in the way of breathing and then the patient gets tired breathing. The respirator does not help at all. The patient must be put on his face, and the foot of the bed raised. There should be a suction apparatus handy, and a special nurse present who does nothing but apply suction to the patient's throat to keep the throat clear. Most of these patients will get along pretty well.

We are trying out a new procedure at the Willard Parker Hospital at the present time. We are trying the effect of thiamin chloride intravenously in massive doses, 20 mg. three times a day. A patient came in this summer who had involvement of both arms and both sternocleidomastoid muscles, which is a usual involvement when both deltoids are affected. The muscles were not gone but were pretty weak. We started giving him thiamin chloride, two doses a day. The afternoon of the next day he jumped out of bed and ran down the hall. We had to catch him and put him back in bed again. He also had 9 mg. of hydrochloride by mouth three times a day.

As to the question of the maximum pressure in the respirator, it is usually negative. This varies a great deal with the child. Some children will cooperate with the respirator better on a positive pressure, while some will do it better on a negative pressure. As a rule, we usually start on a negative pressure and experiment a little bit by shifting back and forth. If we had a respirator available, we put in it a bulbar patient who was expected to die anyway, just to see what help it would give him. Once in a great while a child seemed to be helped by it, but usually if there was bulbar involvement, the respirator did more damage than good.

DISCUSSION

CHAIRMAN HOYNE opened the round table with a discussion on the essential factors of quarantine.

DR. TOP.—Clinical tests determine the release from quarantines. Bacteria are still present in the throats of those convalescing from scarlet fever after twenty-eight days. Scarlet fever is milder on the whole than it used to be; therefore it is right to change the quarantine. Children released from the home are less frequently found to be the source of repeat cases than those released from the hospital. The

reason seems to be rather obvious. The hospital patient has shared an exposure to strains of *Streptococcus haemolyticus* other than the original strain causing the disease. The infecting case rate is 3.2 per cent, while the return rate is 4.1 per cent.

Wage earners do not frequently transmit the disease when allowed to continue their work. This gives evidence of fairly general cooperation on the part of parents and wage earners who must continue their work. Food-handlers are still quarantined. In the case of measles, young children should be strictly isolated, even though the contagion is lost soon after complete eruption.

DR. STIMSON.—Adults average three contagious diseases per person, one contagious disease for each six children per year. Each year 1,500,000 cases are reported out of a probable 8,000,000 cases. Smallpox and diphtheria are nearly all reported. Only one out of ten cases of pertussis is reported. Whooping cough, like the virus diseases, is very contagious. Regulation of bacterial diseases should be very strict, whereas virus diseases are not carried through the open air. I have noted that if a nasal discharge is present in scarlet fever, it is a fairly good sign of impending complications.

Isolation during seasons when common colds are epidemic should be more rigid or of longer duration than in seasons when the common colds are less prevalent. Rural dwellers are more susceptible than city dwellers to contagion.

Relapses in hospital cases are surely due to exchange of different strains of the bacteria. Every hospital case should be isolated from every other hospital case. The isolation of carriers is rather impractical, while the immunization of contacts is rather effectual. In 1922 in the Willard Parker Hospital, there was a 22 per cent mortality among cases of measles, due of course to secondary infections.

CHAIRMAN HOYNE.—The quarantine period in Chicago is somewhat different from that in Detroit and New York. We date our onset from the beginning of the quarantine or the day the disease was reported. Our return cases nearly always have the same type of streptococcic infection that the original case had on entering the hospital.

In Chicago, I have never seen a repeat case of poliomyelitis after return from the hospital of the infected patient. It is questionable if isolation is at all necessary. In 1933, out of 14,000 patients with scarlet fever, the return cases were more severe than the original infection in the homes. The milder cases of scarlet fever seem to be more the cause of return cases than are the severe cases of which all persons are rightly afraid.

DR. J. V. GREENEBAUM, CINCINNATI.—Why are cases in patients over 15 years of age less dangerous?

DR. A. GOLDBLOOM, MONTREAL.—Please clarify the use of the terms "return case" and "infecting case."

DR. H. C. GRAHAM, TULSA.—Have any of you seen scarlet fever in individuals that have a negative Dick test? Why not use toxin if the patient is left at home the same as if he were taken to the hospital?

DR. H. L. DWYER, KANSAS CITY, MO.—Can the quarantine be omitted if the patient has received serum?

DR. BERRY, BIRMINGHAM, AND DR. GRAHAM.—Is any consideration to be shown contacts that have received toxin or serum? Can they go to school?

DR. J. S. WALL, WASHINGTON, D. C.—Why quarantine measles at all?

DR. TOP.—What is the effect of school closure on scarlet fever epidemics?

DR. R. C. HOOD, CHILDREN'S BUREAU, WASHINGTON, D. C.—What do you pediatricians say should be done with severely acute cases of poliomyelitis when the hospital refuses to admit them? Is there not grave danger of respiratory paralysis in any one of these cases?

DR. M. L. BLATT, CHICAGO.—Should there be authority to terminate quarantine by the result of throat cultures and in whooping cough on cultures or cough plates?

DR. J. L. GEDGOUD, CHICAGO.—Do cases immunized by toxin injections still have bacterial pharyngitis?

DR. F. R. JANNEY, WAUWATOSA, WIS.—In case of staphylococcus osteomyelitis is there any confirmation found in gastric washings?

CHAIRMAN HOYNE.—No.

DR. A. D. KAISER, ROCHESTER, N. Y.—Is there no danger in failing to quarantine poliomyelitis?

DR. STIMSON.—We ought to be thinking of it, but not necessarily changing the quarantine as yet.

DR. GREENEBAUM.—Are there more return cases caused by the younger patients because they return to school, than by the older patients, who return to work?

DR. GOLDBLOOM.—I still do not see the difference between return case and infecting case.

DR. TOP.—During the incidence of virus diseases, chiefly upper respiratory diseases, complications in scarlet fever cases are definitely in excess. The severity of scarlet fever is on the decline, but if the severity changes, we may have to change our attitude toward any changes in quarantine restrictions.

As to the isolation of every hospital case, I find that isolation through glass cubicles is fairly satisfactory, but children do not all do well when alone.

We allow quarantine in hospital cases to be figured from the onset of the disease. In the home, cases are quarantined from the date of report, thus favoring early report by the physician. Home treated cases are instructed daily as to the danger of contact infections.

DR. DWYER.—I think every patient with poliomyelitis should be in a hospital, and every hospital ought to accept a case of poliomyelitis. They ought to be in a hospital because you don't know what is going to happen to some of them. Some cases start out very mild at the onset, and overnight develop a marked paralysis and have to be placed in a respirator.

DR. STIMSON.—I simply mean to warn of the danger of moving a poliomyelitis patient to the hospital in an improper manner, without due caution, for this may cost him his life or may cause him to become a permanent or hopeless cripple, whereas much less than hospital care at home might afford a good result. It would be rather good psychology to take the child away from the family if they are able to pay for the hospital care. Give them what they feel they would like to have, and at the same time protect yourself and protect the child.

If the hospital is near and the move easy, placing a child in a hospital may be desirable, but a long, hard ride is definitely harmful; and it is only in a very small percentage of cases that a respirator is needed and beneficial.

If, for instance, there is a poliomyelitis epidemic and a child in that vicinity is found to have an upper respiratory infection or a little alimentary disturbance, the patient should be treated as though he had infantile paralysis; he should be kept absolutely quiet with a very minimum of handling, not moved to a hospital, not allowed out of bed, not handled by anybody when unnecessary.

The child's chances of avoiding any evidence of paralysis are much better if that is the procedure. On the other hand, we hear time and again of people who have these preliminary manifestations, who keep on their jobs, work, or go to school, or whose parent gets panicky and drive them in an automobile 150 miles from a farm in the country to a hospital in the city, where the child develops a paralysis. The child must be left on that farm unless there is a respiratory involvement.

The second point is Dr. Wall's question about measles. In a symposium on viruses published recently, there is an excellent chapter on measles. They offer pretty good evidence that the complications of measles do not affect in any way the infectivity of measles itself; that is, if a patient with measles has a streptococcal infection, the measles is no more communicable, certainly not after five days' duration of the rash. Measles has been transmitted by transfusion, the blood having been taken from people incubating the disease, and certainly during the last half of the incubation period and during the early stage of the incubation period.

As regards the isolation period of chicken pox, the School Physicians Association of New York City allows children to go back to school even with scabs on them, provided they have had thorough baths and shampoos. It seems to be very important that they have shampoos.

As regards the value of the negative Dick test, at the Willard Parker Hospital up to Jan. 1, 1937, 2½ per cent of the student nurses came down with scarlet fever during their three months' course in the Willard Parker Hospital, although they were all supposed to have negative Dick tests on arrival. Then the Willard Parker Hospital began doing its own Dick tests, and found that a very considerable proportion of these girls really did have positive Dick tests. Then we began immunizing them ourselves. Since Jan. 1, 1937, we have not had one single case of scarlet fever in a Dick-negative person among 1,200 nurses, although we have had seventeen cases in Dick-positive physicians and graduate nurses. Now, whether some of those girls have had streptococcal sore throats due to scarlet fever, we cannot tell; some of them have been ill just the way some of the other people in the hospital have been sick: whether some of their illnesses have been due to the scarlet fever streptococcus, I cannot say.

In regard to the immunizing dose, at Willard Parker we begin with only 100 skin test doses, and double the dose every week, giving the patients about ten doses before they are satisfied, and the result is that the last three or four doses are 50/1000 skin test doses or thereabouts. The result is that the total received something over 200/1000 skin test doses.

DR. ASHMUN.—It is our impression at the Willard Parker Hospital and probably of most of us that the recognized case of poliomyelitis is the unusual case, that there are a great many more people infected with poliomyelitis than go to a hospital with it, and that isolating the one patient does not accomplish very much.

The average general hospital should admit poliomyelitis patients to their wards because, with one exception, no physician or nurse has ever been known to get

poliomyelitis from his or her patient. Certainly that has been the case at Willard Parker. Last summer a student nurse was caring for poliomyelitis patients. She left on Sunday, and on Tuesday she thought she had early symptoms of what might be poliomyelitis. On Thursday she had definite evidences, and on Friday she had all the manifestations and died on Saturday. That is the only incidence we have had of a nurse or a physician contracting poliomyelitis, quite obviously a result of contact with the patient.

Those of us who have seen a lot of poliomyelitis are very firmly convinced that most patients do better at home than in a hospital unless they have evidence of respiratory involvement. Even the move from the home to the hospital may do the patient damage, and we feel quite strongly that patients should have the least possible handling from the earliest possible evidence of trouble.

Academy News

ANNUAL MEETING, REGION IV OF THE AMERICAN ACADEMY OF PEDIATRICS

The annual meeting of Region IV of the American Academy of Pediatrics was held in San Francisco, May 1, 2, and 3, 1941. Three-fourths of the members of the Region were in attendance.

The first day was devoted to clinics which were held in the morning at the University of California Medical School under the direction of Dr. Francis Scott Smyth and staff. In the afternoon similar clinics were held at Stanford University Medical School under the direction of Dr. Harold K. Faber and staff. The following two days were devoted to a scientific program.

Business Meeting of Region IV

It was voted to appropriate $500 from the funds of the Region for the purpose of chemotherapeutic drugs for the treatment of British children. It was made possible to send 200,000 5-gr. sulfanilamide tablets and 26,000 ½-Gm. sulfathiazole tablets to the British Pediatric Society in care of the president, Dr. W. J. Wyllie, at the Hospital for Sick Children, Great Ormond Street, London.

The following resolution was also adopted by the Region at this meeting and was sent to the Surgeon General of the United States Army, the Surgeon General of the United States Navy, and to the Chairman of the Committee on National Defense:

"Whereas pediatricians are eager to serve in the National Defense Program to the best interests of the Federal Government, and

"Whereas the pediatrician occupies an unusually favorable position, by virtue of his training and experience, with regard to the management of nutritional problems and the problems concerned with sanitation and contagious disease control, therefore

"Be it resolved by Region IV of the American Academy of Pediatrics in session in San Francisco, Calif., May 1, 2, and 3, 1941, that the Surgeon General of the United States Army (the Surgeon General of the United States Navy, the Chairman of the Committee on National Defense) be respectfully urged to outline the objectives to be fulfilled in the Army (Navy, National Defense) by the pediatricians of the nation individually and collectively, and that furthermore

"In the formulation of such a policy for the service, both public and private, of pediatricians to the nation, that their exceptional training in nutritional problems, the care of the contagious diseases, and the supervision of child welfare in the nation be given especial consideration."

In response to this resolution favorable replies were received from all three agencies.

The Regional Meeting terminated in an enjoyable dinner dance and floor show.

TENTH ANNUAL MEETING, REGION III OF THE AMERICAN ACADEMY OF PEDIATRICS

Meeting of State Chairmen, Region III

The annual meeting of State Chairmen for Region III was held on May 16, 1941, in Chicago, Ill. The meeting was called to order by Dr. A. Graeme Mitchell, Regional Chairman.

Dr. Edward A. Wishropp presented a report.

The tenth annual meeting of Region III of the American Academy of Pediatrics was held at the Palmer House, Chicago, Ill., on May 15, 16, and 17, 1941.

The registration, which totaled 664, was made up as follows: members, 240; nonmembers, 214; scientific exhibitors, 8; commercial exhibitors, 87; and ladies, 115.

There were thirty-eight exhibit spaces occupied by thirty-four commercial exhibitors and ten spaces devoted to scientific exhibits.

At the luncheon meeting on Thursday, May 15, honoring Dr. Isaac Abt, Dr. Joseph Brennemann, and Dr. T. B. Cooley, there were 311 persons present.

The cocktail party and banquet Friday evening were attended by 403 persons.

One hundred and fifteen ladies attended the complimentary luncheon and style show at Marshall Field & Company as guests of the Region.

Business Meeting of Region III

After thorough discussion it was decided to continue the Committee on Arrangements in full charge of the annual meetings of the Region. The plan has functioned efficiently and provides for a continuing committee to handle the regional meeting.

It was moved by Dr. Wiley Jones and seconded by Dr. George Munns that the present treasurer continue to handle the funds of the Region for the ensuing year. The motion was passed unanimously.

The treasurer was empowered to arrange for an annual audit of the Regional funds as of July 1, 1941, by a Certified Public Accountant, the expense of the auditing to be paid from the funds of the Region.

The Committee on Arrangements for the next Annual Meeting was appointed as follows:

Dr. E. A. Wishropp, Chairman
Dr. W. J. Scott
Dr. W. S. Nolting
Dr. J. Hugh Lewis
Dr. Edgar E. Martmer, Ex-Officio

Cleveland, Ohio, was selected for the Eleventh Annual Regional Meeting, the time of the meeting to be decided by the Committee on Arrangements.

Dr. Henry Gerstenberger was appointed Local Chairman of the Program Committee and Dr. John Toomey, Local Chairman on Arrangements.

The policy of charging a registration fee sufficient to cover the cost of a luncheon and banquet ticket was discussed, and the policy of charging a $5 registration fee was officially approved.

Obituaries

Word has been received of the death of Sir George Frederic Still, Professor Emeritus of Diseases of Children at King's College, London, and Physician Extraordinary to the King, at his home, Harnham Croft, Salisbury, England. He was Honorary Chief of Staff at Great Ormond Street Hospital for Sick Children in London. He had visited in this country and was an honorary member of the American Pediatric Society since 1903. His professorship at King's College was the first in pediatrics in Great Britain. He was a Fellow of the Royal College of Physicians.

Dr. John P. Crozier Griffith, of Philadelphia, died July 28, 1941, at his home, Devon, Pa., at the age of 85 years. Dr. Griffith was one of the outstanding pediatricians of this country and belonged to a group which had most to do with the founding of pediatrics as a specialty. He had been in the field of medicine for more than fifty years. He was Professor Emeritus of Pediatrics at the University of Pennsylvania, having been a member of the faculty since 1889.

He was the author of several books and many articles. His outstanding book was a textbook on pediatrics, first in a large edition and later in a single volume edition, being associated with the late Dr. A. Graeme Mitchell of Cincinnati. He was a member of the American Pediatric Society and was its president in 1903. He was also a member of the Association of American Physicians, serving as treasurer from 1900 to 1918.

He had retired from active practice and teaching for several years. His death removes another important figure from American pediatrics.

Dr. Paul B. Bender, a prominent pediatrician of Philadelphia, died on May 13, 1941, at his home. He was 52 years old. He was a Fellow of the American Academy of Pediatrics.